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EDITORIAL

THE EMPIRE CONFERENCE

It is claimed for a classical education that it teaches the pupil to learn, and we have heard it suggested that tuberculosis might well be taken as a classic in medicine, because the student with a good grounding in this subject must appreciate the principles of general medicine and surgery. A knowledge of tuberculosis necessitates the study of toxæmia, temperature, symptoms due to disease of lungs, kidney, intestines, bones, joints and indeed of every organ in the body. Lastly the student who has thought deeply on the problem must have an understanding of human nature, a subject so vital to every physician, but one which is still sadly neglected in the medical curriculum.

Happiness is the essence of life, and it is largely the ability to accept one's lot with contentment. It is not to be bought with money, nor is it a matter of health. It is largely a matter of temperament, and it is, in fact, the art of life. We cannot all be first-class artists, but we can improve with help. Those of us who have seen the wonderful work of St. Dunstan's must have marvelled at the amount of happiness which has been brought to the blind. The same problem is present in tuberculosis, and however skilful a surgeon may be at treating a tuberculous knee, or with whatever success a physician may rest the lung by pneumothorax, it profits the patient little if he is left unsettled, discontented, and with a grievance against the world.

Now tuberculosis differs from most afflictions in that the sufferer has periods of good health during which he can work and play and lead the same life as friends of his own age, but these bouts of health are followed

by relapses. It is a disease of youth, and with characteristic optimism the patient regards himself as cured as soon as he recovers from a relapse, but the repeated shattering of his expectations gradually kills hope and leads to that state of mind which the physician should do everything possible to prevent.

There are as many factors in the problem of tuberculosis as there are pieces to a jig-saw puzzle, but they may be divided into two main groups. First, methods intended to cure the disease, or if this is impossible to procure as much improvement as possible. Secondly, the after-care of the patient.

For the purpose of treatment the hospital and sanatorium with medical and surgical procedures to combat various pathological conditions produced by the disease are of first importance, and let us not forget that modern treatment meets with so much success that many patients are completely cured and able to return to normal lives without restrictions. An institution like the King Edward VII. Sanatorium, Midhurst, is not intended for patients in the acute febrile stages of the disease for this is the function of the hospital. When he has recovered from this acute phase he can obtain at Midhurst treatment which often leads to a cure. This sanatorium has expanded not only by providing facilities for major surgical operations, but by keeping up-to-date in all directions so that it maintains its claim to be a model sanatorium without becoming a vast institution.

Many a patient leaves the sanatorium cured, some few are worse and live only a short time, but a considerable proportion leave with the disease arrested, though not cured. These patients may eventually become cured, and usually have many years of active life provided they are protected; but they are not able to hold their own in the ordinary markets of the world, and even if they do for a time manage to hold a full job they cannot stay the course. It is for this type of patient that after-care is so urgently needed. Some of them want tiding over a few years only and then become fit to take their place in the outside world, but many require protection all through their lives. It is in its dealing with this aspect of the tuberculosis problem that Papworth Village Settlement is chiefly known, but one must bear in mind that before sanatorium treatment is finished the patient often becomes fit for light work under protected conditions. At Papworth employment can be found for those who can work for no more than two hours a day, and so one can appreciate the value of having a sanatorium in connection with the Colony. Not only is there a sanatorium

but a hospital side, where patients who are febrile at the beginning of their illness or as a result of relapse can receive treatment. The new surgical block, staffed by experienced surgeons, is now in full swing, so that a patient may receive the latest and best surgical treatment. The Settlement is, in fact, a colony where patients in all stages of the disease can be treated, but because its fame largely depends on its after-care work it rightly takes the lead in promoting the Empire Conference on After-care.

We hope to show our friends from overseas what is being done in this country. They will find that Papworth succeeds in making the patient feel he is independent and equal to his fellow-citizens. It is not run on lines of sympathy or pity or still less on patronage. The settler works as others work, his goods are sold in the open market, he earns a wage, and a good one, as a result of his work. He has self-respect, he can be on committees dealing with the entertainments of the Colony, the cinema, theatricals, games, and other recreations. There is no question of " abandon hope all ye who enter here," hope is revived and the one impression a visitor carries away from Papworth is the happiness of its settlers.

It has been stated that of those patients who leave a sanatorium still needing care only a small proportion will consent to become settlers. This is one of those dangerous half-truths, for it is financial considerations that limit the number of settlers. And when one considers the vast sums of money spent by the State on tuberculosis and the unquestionable saving by obtaining useful work from the settlers, quite apart from what some may regard as the sentimental wish to make them happy, one must admit that those who have so generously subscribed to the Settlement have every right to feel pride at the far-seeing policy which induced them to support the scheme.

One should not suppose, however, that this Settlement and others which have copied it are sufficient to deal with the problem of after-care. In this country we have workers, many voluntary, who devote their lives to this problem and aid in many ways the chronic consumptive who needs protection. To them all credit is due, and we hope that all aspects of the problem will be fully discussed at the Conference.

We have something to show our visitors, but we hope to learn much from them. Interchange of ideas is the object of the Conference. What, for example, is the effect of the extremes of heat and cold as in Canada? What of the climate of India? South Africa has a reputation which attracts many consumptives, but it is a vast country: what can our South

African colleagues tell us about the country? New Zealand has a very low tuberculosis death rate and so has Australia: what of these countries? Above all, what can our overseas friends tell us about the psychology of their people? We have no doubt that we shall gain from them many hints, and we hope that in return they will take back with them one or two new ideas on the problem of after-care.

EMPIRE CONFERENCE ON THE CARE AND AFTER-CARE OF THE TUBERCULOUS

UNDER the auspices of the Overseas League and the Papworth Village Settlement an Empire Conference will be held in London, under the Presidency of the Marquess of Willingdon, from May 3 to May 8, 1937, on the Care and After-care of the Tuberculous. The Conference, which has the highest medical patronage, is being called as among the many thousands of visitors in London from the Dominions and Colonies for the Coronation there will be a large number of medical men and women and others interested in tuberculosis. In view of the success of Papworth Village Settlement, not only in arresting tuberculosis, but in successfully employing the ex-patient in productive industry, there appears to be an urgent need for the study of similar methods throughout the Empire.

There is also the Imperial obligation imposed upon the British Dominions and Colonial Governments, and their enlightened subjects, to combine in protecting from the scourge of tuberculosis the millions of pastoral and agricultural workers committed to their charge. These simple peoples, whose voices are seldom audible, but whose work helps to build up the prosperity of the Empire, exhibit a marked natural tendency to tuberculosis, accentuated by extreme poverty, bad housing, inadequate nutrition, and a medieval type of domestic hygiene. They constitute a problem which should appeal to us all.

The following preliminary programme shows that the Conference speakers will include a number of distinguished medical men. To these there will be added others from overseas. The Conference will therefore provide an unique opportunity for exchange of views.

In connection with the Conference, which will be held in the newly erected Conference Hall at Overseas House, St. James's, London, visits will be organised to Papworth Village Settlement in Cambridgeshire for those who wish to see something of the work that is being done there.

Overseas League Members and others who desire, during their stay in London for the Coronation, to take part in this Conference, are invited to complete the enclosed application form and return it as soon as possible to: The Secretary, Empire Conference on the Care and After-care of the Tuberculous, Overseas House, St. James's, London, S.W. 1.

Correspondents having friends who would like to attend are recommended to suggest to them that they should notify the secretary by postcard.

CONFERENCE ON THE CARE AND AFTER-CARE OF THE TUBERCULOUS.

PROGRAMME.

First Session.

Monday, May 3, 1937. Morning, 10.30 a.m. to 12.45 p.m.

Chairman : The Marquess of Willingdon, G.C.S.I., G.C.M.G., G.C.I.E.
Subject : " The Fight against Tuberculosis throughout the Empire."
Speakers : The Rt. Hon. Sir Kingsley Wood, M.P. (Minister of Health); Professor S. Lyle Cummins, C.B., C.M.G., LL.D., M.D.; followed by representatives from the Dominions, Colonies and Protectorates.

Second Session.

Monday, May 3, 1937. Afternoon, 3 p.m. to 4.30 p.m.

Chairman : The Rt. Hon. Lord Dawson of Penn, G.C.V.O., K.C.B., M.D., F.R.C.P.
Subject : " The Fight against Tuberculosis throughout the Empire."
Speakers : Representatives from the Dominions, Colonies and Protectorates.

Third Session.

Tuesday, May 4, 1937. Morning, 10.30 a.m. to 12.45 p.m.

Chairman : The Rt. Hon. Lord Horder, K.C.V.O., M.D., F.R.C.P.
Subject : " The After-care of the Tuberculous throughout the Empire"
Speakers : Sir Pendrill Varrier-Jones, M.A., F.R.C.P.; Dr. B. A. Dormer (representative of the Medical Services of the Union of South Africa); followed by representatives from the Dominions, Colonies and Protectorates.

Fourth Session.

Tuesday, May 4, 1937. Afternoon, 3 p.m. to 4.30 p.m.

Chairman : Professor S. Lyle Cummins, C.B., C.M.G., LL.D., M.D.
Subject : " The After-care of the Tuberculous throughout the Empire."
Speakers : Representatives from the Dominions, Colonies and Protectorates.

Fifth Session.

Wednesday, May 5, 1937. Morning, 10.30 a.m. to 12.45 p.m.

Chairman : The Viscount Goschen, G.C.S.I., G.C.I.E. (Chairman of the Overseas League).
Subject : " The Native Races and Tuberculosis throughout the Empire."
Speakers : Lieut.-Colonel G. G. Jolly, I.M.S. (representative of the East India Association); followed by representatives from the Dominions, Colonies and Protectorates.

Sixth Session.

Wednesday, May 5, 1937. Afternoon, 3 p.m. to 4.30 p.m.

Chairman : The Hon. Sir Firozkhan Noon (High Commissioner for India).

Subject : "The Native Races and Tuberculosis throughout the Empire."

Speakers : Dr. P. V. G. Benjamin (Mission Sanatorium, Madanapalle); followed by representatives from the Dominions, Colonies and Protectorates.

On Friday, May 7, members will be welcomed at Papworth.

Special motor coaches for Papworth will leave Overseas House at 9 a.m. For members using private cars, the route indicated by the accompanying plan is recommended.

The Marquess of Willingdon attended an informal meeting held on January 22, 1937, at Overseas House, St. James's, when Sir Pendrill Varrier-Jones, F.R.C.P. (Medical Director of Papworth Village Settlement), explained the reasons for convening the Empire Conference on the Care and After-care of the Tuberculous to be held at Overseas House, London, from May 3 to May 5 this year. Lord Willingdon is President of the Conference, and the Patrons include H.R.H. the Duke of Kent, five members of the Government, the Viceroy of India, the Governors-General of Canada, Australia, and South Africa, the Governor of Newfoundland, and many prominent representatives of the countries of the British Empire. The list of Vice-Presidents is headed by Viscount Dawson of Penn and Lord Horder, and the Conference is being organised jointly by the Overseas League and Papworth Village Settlement.

Sir Pendrill Varrier-Jones pointed out that the Coronation afforded an unrivalled opportunity for the gathering together of representatives from the Dominions and Colonies, and that there was much to be gained from a Conference of the kind proposed. The response to the proposal had been both immediate and enthusiastic; for while tuberculosis was on the decline in Great Britain, in various parts of the Empire this was not the case.

On the overwhelmingly important subject of immunity, he felt that the advice of overseas representatives would be invaluable. In many cases they were in a position to study the impact of tuberculosis upon peoples with no acquired resistance, and the results of observation and study in such conditions would, he felt, be of material assistance here.

Similarly, experience in Great Britain would provide means of meeting many of the problems of tuberculosis arising in the Dominions and Colonies. He was therefore of opinion that an exchange of experience was not only desirable but essential.

In the case of tuberculosis, the speaker said, Great Britain had a special responsibility towards the Empire peoples. It was not only the Great

White Scourge: it was a White Man's Disease—one of those which had been introduced by the white man to races which had hitherto been free from it. To spare no effort in order to conquer it was therefore part of our Imperial responsibility and could not be evaded.

More and more it was being recognised that tuberculosis was a disease requiring to be combated on every front—social and economic as well as medical. Therefore in the coming discussion he hoped that emphasis would be laid not only upon clinical signs, X-ray indications and so on, but also upon living and working conditions. Unless the victim of tuberculosis were afforded an opportunity of working and earning in a suitable environment and under suitable conditions, little progress, he felt, could be made in stamping out the spread of infection; and furthermore infinite risk of general disaffection would arise.

Here was yet another reason for urgency. Tuberculosis was productive of mental as well as physical disease. The victim, deprived of work, deprived therefore of the means of livelihood, was in extreme danger of contamination by subversive influences; and it was a question whether in the long run these were not even more dangerous to public security than the disease itself. In the 1935 Papworth Report this vitally important aspect of the matter had been dealt with; and the speaker quoted an apposite extract from a recent issue of *The Lancet*:

" The nucleus of the whole problem lies in the mode of dealing with aggressive impulses. If these are coupled with a pleasure in constructive abilities, the result is productive work: an example of this may be seen in the gigantic undertakings in Russia to-day. If the individual cannot find satisfaction in constructive activity, for instance, if he is unemployed or is put to an uncongenial occupation, the necessary condition for the binding of the aggressive impulse to cultural ends is lacking and there is a risk of a breakdown of social relations. The political analogy is revolution, if the aggression is kept within the frontiers, or war if the aggression is projected outwards. This is the reason why in the case of an individual faced with an impending crisis it is so important that his energies should to the last moment be given a constructive outlet."

" For every reason, therefore," Sir Pendrill concluded, " we feel that this Conference is necessary. First, because even now tuberculosis is killing thousands of our people every week and creating widespread privation and distress. Second, because it is the white races which have brought this disease to other races, who therefore need and deserve all the help we can give them in combating it. Third, because an exchange of experience between medical men in different parts of the Empire must increase our mutual store of knowledge; and, last but not least, because the absence of

comprehensive and successful measures to fight tuberculosis may lead to consequences of the gravest urgency in the political field. It is our sincere and earnest, hope, therefore, that this Conference may succeed in bringing us nearer to the control and conquest of a disease which is as yet unconquered and which still threatens the health, the resources, and the future of our Empire."

Professor S. Lyle Cummins said that the peoples of our Empire fall into two groups: those of European, chiefly British origin, and those constituted by the original populations of the territories which comprise the Empire. In the case of the former, the medical men, trained in their own Universities in the Dominions or in the Universities of Great Britain, are keenly alive to the importance of the tuberculosis problem, and our Dominions can show examples of sanatorium development deserving of the highest admiration.

For these Overseas communities of European stock the value of such a Conference as is now contemplated is inestimable, enabling them to study at first hand developments such as the Papworth Industrial Settlement, organised on such a wonderful system by the late Sir German Sims Woodhead and Sir Pendrill Varrier-Jones, as well as other great sanatorium developments in this country; and they do not only come to learn, but also to impart to us the many new ideas that they themselves are developing in their own territories. But when we come to think of the other section of our Imperial populations, the aboriginal races, still forming a great majority in many of our Overseas Dominions, Colonies and Protectorates, we are faced with the tuberculosis problem in a very acute as well as a very important form. These populations, native to the territories in which they reside, had originally lived a much more primitive life than is possible for them today. Under these more or less primitive conditions to which they had been for generations accustomed, they enjoyed relative freedom from endemic tuberculosis, chiefly because the conditions of life involved no confinement to over-crowded industrial communities and little or no contact with outsiders from industrialised Europe. These conditions have altered or are altering today. The European cannot but bring with him his science and his industry when he assumes the responsibility for developing the territories of our Overseas Empire, nor can he, unhappily, avoid bringing with him the germs of the endemic diseases which have established themselves for long centuries in the crowded civilisations of Europe. Thus he introduces into the older cultures which he invades two new elements—the germs which he himself has come to tolerate, but which may be fatal to "virgin soil"; and the industrial conditions to which he is accustomed, but which are foreign to those amongst whom his new work is placed. The effects

of these new factors are often tragic for these primitive peoples, but can be much ameliorated if along with them are introduced the scientific methods applied for the protection of our own home communities under similar conditions.

It should be a source of pride to our Empire to note how some of our great industrial groups working amongst primitive communities face up to the expense and trouble which they feel that they should impose on themselves in the interests of the employees. Such an organisation as the Chamber of Mines of Johannesburg is a splendid example in this respect, and they seem to have for many years known and appreciated the fact that "economic morality" pays: a discovery which Mr. Roosevelt has recently enunciated in the United States. They have realised that a healthy and contented body of workers is well worth while, and that sums of money expended in prevention of disease may be saved in industrial compensation; but while this is so true of some of our greatest Overseas industrial developments it is not universally true, nor have we arrived at any final conclusion as to the best method of prevention and cure. Such a Conference as that now contemplated appears to me an essential step towards discussing and, perhaps, comprehending better the intricate problems of the prevention and cure of tuberculosis in those native populations of our Dominions and Dependencies found to be intensely susceptible to tuberculosis when brought under industrial conditions. It is for this reason that I have welcomed from the first the conception evolved by Sir Pendrill Varrier-Jones of attempting to bring about the Imperial Tuberculosis Conference, which is now to become a reality.

RECEPTION AT THE BROMPTON HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST.

The Brompton Hospital has arranged a reception on May 6 for the Delegates to the Conference. The hospital will be open to inspection, demonstrations of the medical and surgical work will be given and tea will be served in the Board Room.

GENERAL ARTICLES

THE THERAPEUTIC POSSIBILITIES OF ARTIFICIAL PLEURAL EFFUSIONS

By GUSTAV MAURER,
M.D.,
Medical Superintendent, Schatzalp Sanatorium, Davos, Switzerland.

In this article I shall attempt to describe the types of collapse treatment in which the artificial pleural effusion, by producing a symphysis between the parietal and visceral pleura, may be beneficial. An exudate, natural or artificial, leads to union of the two pleural surfaces when they are allowed to touch one another during the phase of reabsorption and organisation.

In three-quarters of the cases of pulmonary tuberculosis the lesions are limited to one or both upper lobes in the earlier stages, and, as a rule, it is only later on that the middle and lower lobes become involved. We can see, therefore, that for our problem it is important to bear in mind this fact: that in the majority of tuberculous patients, when they first present themselves for treatment, the infiltration or cavities are chiefly in the upper lobes, and that when once the process has spread more and more into the lung bases it is usually too late for any therapy.

To the French school—Besançon, Dumarest, Rist and Sergent—belongs the credit of first pointing out the great frequency in which disease is confined to one lobe. Since the introduction of radiography there has been a tendency more and more to depend on the X-ray film in the diagnosis of pulmonary tuberculosis; so much so that we are in danger of losing sight of this fact—*i.e.*, unilobar tuberculosis. Now, it is unfortunate that the several margins of the lung lobes are in oblique and not horizontal planes. By the usual description into upper, middle, and basal regions we are unable to give an accurate idea in which lobe a lesion is situated. Briefly, overlapping of the lobes makes diagnosis of the seat of lesion difficult in the X-ray picture. Here stereoscopic radiography or, better still, tomography may help.

A patient suffering from tuberculosis in one lung only, serious enough to require collapse therapy, may be treated by a unilateral pneumothorax, phrenicectomy, thoracoplasty, and, exceptionally, by apicolysis or the new treatment, extrapleural pneumothorax. But only too often we see the tubercular process start on both sides. What shall we do with these cases? In the last twenty years more and more attempts at bilateral collapse therapy have been made. It is my aim, firstly, to show how poor are the results of bilateral pneumothorax, pneumothorax with contralateral phrenicectomy, apicolysis or thoracoplasty; secondly, I intend to show why bilateral collapse so often fails; and, lastly, how it may be rendered more successful.

According to the most reliable statistics, pulmonary disease is arrested in 30 to 60 per cent. of cases after unilateral pneumothorax treatment. The great difference in the results is dependent upon the care with which cases are selected for treatment, social conditions, and how far it is possible to convert incomplete into complete pneumothoraces.

The most striking statistics of bilateral pneumothorax treatment in many hundreds of cases come from the sanatoria of Beelitz. In only 12 per cent. was the disease permanently arrested. Are we to be satisfied with 12 per cent. cure, even when we take into consideration the fact that a double-sided lung tuberculosis has always a worse prognosis than a unilateral process, and that a double pneumothorax is sometimes induced as a last hope? I am wondering whether the very competent physicians of bygone days would not have shown, without pneumothorax treatment at all, better results in bilateral tuberculosis.

Now, why does bilateral collapse therapy fail so often? Pneumothorax treatment is more effective the more the collapse and immobilisation of the lung. The maximum effect is obtained when the pressure in the pneumothorax cavity is round about zero. It is quite clear that the patient will die of suffocation if we give refills with intrathoracic end pressures of zero on both sides. We cannot, therefore, in double pneumothorax cases obtain an ideal effective collapse of either lung. Besides this, immobilisation of the diseased organs is not at all possible, for the patient must breathe.

Gas analyses have shown that in unilateral pneumothorax cases the blood is always sufficiently oxygenated, but never in bilateral pneumothorax. This oxygen deficiency in the blood seems to be an explanation of the fact that the bilateral pneumothorax patient often has so much difficulty in improving in general health. The limited therapeutic value of the bilateral pneumothorax is not its only disadvantage. When a pleural effusion occurs on one side—or worse, on both sides—it becomes dangerous. The patient may die of asphyxia because the movements of the partially

collapsed lungs are reduced in a short time by thickening of the covering visceral pleuræ as well as by the direct mechanical action of the exudate.

It is astonishing how much more effective and safer the bilateral pneumothorax becomes if we fix the sound areas of the lung to the chest wall in such a way that the diseased areas only are collapsed. In all cases of purely lobar tuberculosis there is a distinct difference in volume between the diseased and normal lobes of a pneumothorax lung. The pathological part shows a diminution in size, due to retraction of its foci and atelectasis. The undiseased lung lobes retain a fairly normal volume. This is "selective pneumothorax." By adjusting the intrapleural pressure suitably we can bring the healthy lung lobe into contact with the chest wall, while the diseased upper lobe remains sufficiently retracted not to touch the chest wall anywhere. This is the stage at which I produce an artificial effusion, which, during the reabsorption and organisation phase, obliterates the lumen between the two pleural surfaces wherever they are in contact—that is to say, all over the healthy parts of the lung. Finally, an air pocket remains only over the diseased retracted areas. We may call this a "lobar pneumothorax."

When this favourable state of affairs has been realised, we can, by the usual refills with an end pressure around zero, entirely collapse and immobilise the diseased lobe without impairing the function of the sound and now adherent lower lobe. The re-expanded and fixed bases will guarantee sufficient gaseous exchange, and even if a pleural effusion does occur in one or both lobar pneumothoraces, we shall not lose a patient from suffocation.

And now a few words as to the technique. The artificial symphysis of healthy lung lobes is produced by injecting 50 per cent. glucose into the pleural cavity. The glucose does not act as a chemical irritant, but as a hypertonic solution—it causes bursting and desquamation of the endothelial cells and a serous exudation from the denuded surfaces. After making the intrapleural injection, enough air is withdrawn to bring the visceral and parietal pleura over the lower lobe into contact, but not enough to expand the diseased and retracted upper lobe. Otherwise it is in danger of becoming also adherent. Constant fluoroscopic control is of course necessary. Thus we can prevent by a small refill any undesirably extensive fixation of the lung. On the other hand, we must avoid too early or too large refills, which might prematurely separate the parts of the pleural surfaces which we wish to adhere. The initial dose of glucose is 50 c.c. Usually this is sufficient. However, I have had cases in which further injections of 100 to 150 c.c. were necessary to produce the desired symphysis. The sensitivity of the pleura varies from case to case. The injections cause slight and transient pain, which is never severe enough to require morphia. There

is always some rise of temperature—the highest observed was 103° F.—but it drops to normal usually in three or four days, at the longest in a couple of weeks. Up to the present I have not observed any severe complication, such as cloudy or purulent exudate, progressive pleural symphysis, or dissemination of the disease in the re-expanded and fixed lobe. The following cases give the indications for the treatment by artificial pleural effusions:

CASE 1.—*Bilateral Apical Pneumothorax.*—This was originally a case of cavernous infiltration of the right upper lobe. No disease could be found in the middle and lower lobe. There was also an infraclavicular infiltration on the left side without signs of destruction. A complete pneumothorax on the right side led to distinct improvement of the focus in the right upper lobe. Owing to the additional strain on the left lung by reason of the pneumothorax on the right side, the infraclavicular infiltration on the left side softened and became cavernous. A double pneumothorax was induced. In the meantime the base of the right lung became adherent by natural means. The pneumothorax on the left side was incomplete owing to adhesions in the region of the apical cavity. These adhesions were cauterised. Thoracoscopy showed the left lower lobe apparently free from disease, and this was confirmed by radiological examination (stereoscopic). The left lower lobe and the healthy base of the upper lobe were made artificially adherent to the chest wall, so that finally a bilateral apical pneumothorax remained.

Until this intervention the patient had been ill for years with positive sputum and a very bad general condition. It was possible to give weekly refills with a definite positive pressure to the bilateral apical pneumothorax without the patient becoming cyanotic. Since the intervention the patient's general condition has improved considerably, the sputum is much less and free from bacilli.

It should be mentioned that general improvement only occurred when it was possible to collapse completely the diseased portions of the lung after the fixation of the sound areas of the lung to the chest wall.

CASE 2.—This case shows that certain dangers accompany bilateral collapse therapy.

A patient suffering from disseminated, infiltrative pulmonary tuberculosis of the left upper lobe, where there was a destructive process, was given a pneumothorax. This was continued for ten months, when she was referred to me for cauterisation of adhesions owing to the presence of a large hanging cavity. There was already a spread in the base of the right upper lobe, with some honeycombed destruction. Cauterisation was performed on November 17, 1933, and there was no reaction. In order that successful collapse treatment could be carried out on the contralateral side, the healthy left lower lobe was first fixed to the chest wall. Accordingly, on February 11, 1934, 20 c.c. of glucose was injected. This was followed by some reaction, with a slight rise of temperature and a small exudate, which disappeared in a few days.

A further 50 c.c. of glucose was injected on March 14, 1934, without causing any rise of temperature, but was accompanied by an exudate larger than that on the first occasion. This, however, soon become resorbed. On March 28, 1934, it was seen that the left lower lobe was sufficiently fixed to the chest wall to allow a pneumothorax to be induced on the right side. The induction was performed without any difficulty, and the patient felt better than is usual in cases of double pneumothorax. As there were only small foci in the right lung, small refills were given, and fixation of the healthy parts of this lung was not considered necessary. Since May, 1934, the patient has remained free from bacilli and there is practically no sputum. The patient's general condition improved in such a striking manner after the induction of the double pneumothorax that in the autumn of 1934 I allowed her to be treated as an out-patient. In January, 1936, she returned to the sanatorium with an exudate which completely filled the left apical pneumothorax cavity. In spite of this exudate her general condition was good, and she was never in any danger. Every time when the exudate was drawn off there was only little reaction. The pneumothorax cavity, which contains 400 c.c. of exudate, was completely emptied.

No active disease could be seen on the right side.

A pleural effusion in a case of unilateral pneumothorax is of little importance, but the same thing in a double pneumothorax can be dangerous. In the case quoted the exudate did the patient little harm, as it was only a partial exudate above the fixed part of the lung. Had the patient a total pneumothorax, the pneumothorax cavity would have been completely filled, as was the partial pneumothorax.

The case shows clearly that collapse treatment of the contralateral side should only be attempted when the other side has been treated, so that it can take over the functions of the contralateral side.

Finally, this case shows that, in cases where the foci are small, only a small pneumothorax should be maintained, and fixation of the healthy lobes should not be attempted if sufficient breathing surface has been obtained by fixation of the lobes on the contralateral side.

Apical Pneumothorax and Phrenic Paralysis of the Contralateral Side.—The combination of pneumothorax and phrenic paralysis on the contralateral side has not often a favourable result in practice. If there is a widespread destructive process in one lung which requires a maximum degree of collapse if the pneumothorax is to be effective, the contralateral side can take over the functional compensation. If, however, diaphragmatic paralysis—either by phrenic crush, phrenicotomy, or extirpation—is performed on this contralateral side, it will bring about a maximun compensatory costal breathing. All foci which lie in the region of this jerky costal breathing will be unfavourably affected by these increased respiratory claims. If diaphragmatic paralysis, with a pneumothorax on the contralateral side, is

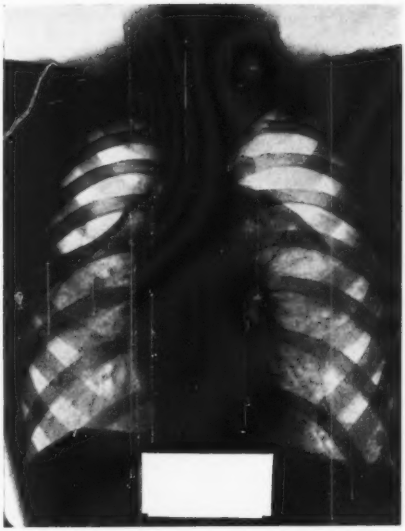

FIG. 1.

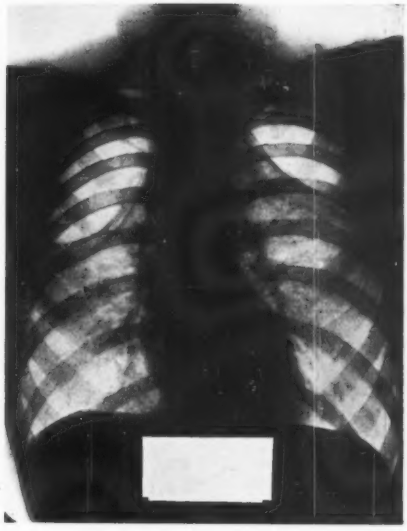

FIG. 2.

FIG. 1.—Before the glucose treatment. The borders of both lobes are plainly seen. The infected upper lobe is less voluminous than the healthy lower lobe, which is almost touching the chest wall (Case 1).

FIG. 2.—After the glucose treatment showing the apical pneumothorax (Case 1).

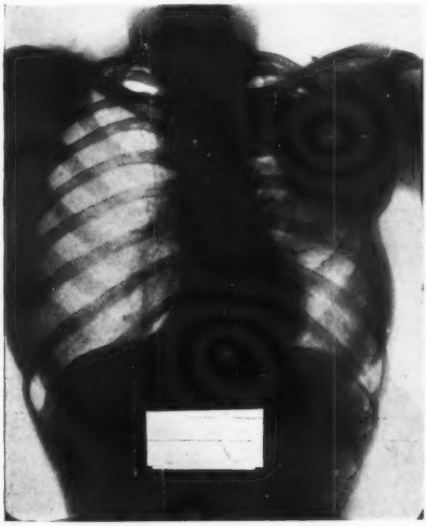

FIG. 3.

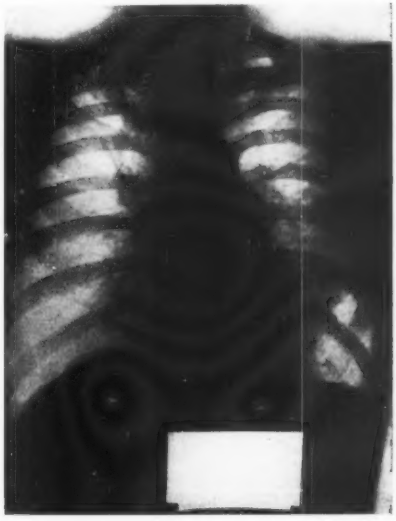

FIG. 4.

FIG. 3.—Shows the described case with artificial basal fixation on the left side and the effusion. Unfortunately the contra-lateral pneumothorax is only faintly seen in the reproduction (Case 2).

FIG. 4.—Shows a pneumothorax on the right side. There is a cavity about the size of a hen's egg in the apex, where there is a broad adhesion. There is a confluent infiltration with honeycombed destruction in the middle part of the left lung (Case 3).

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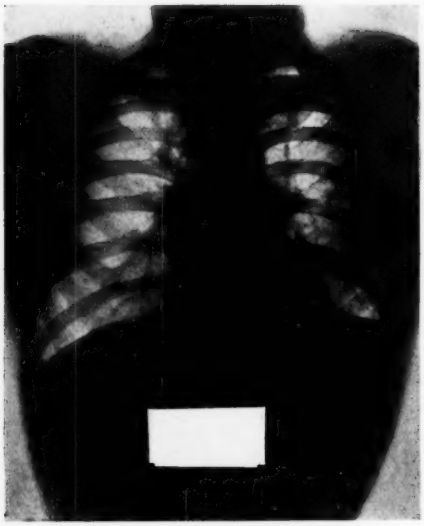

FIG. 5.

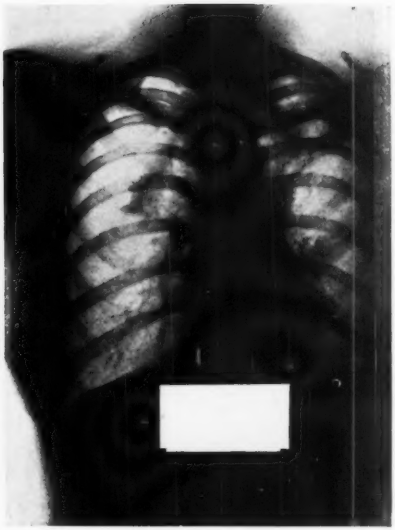

FIG. 6.

FIG. 5.—Shows the right middle and lower lobes fixed, and the pneumothorax is confined to a pocket over the right upper lobe. Before the glucose treatment the apical adhesion was freed. The cavity is no longer visible. On the left side it is noticed that the diaphragm is fairly high after a phrenic crush. The middle parts of the left lung are already clearing up (Case 3).

FIG. 6.—Shows a selective pneumothorax on the right side. The upper lobe is retracted, the middle and lower lobes are almost touching the chest wall. On the left side there is a large apical cavity, and the diaphragm is high after a phrenic extirpation (Case 4).

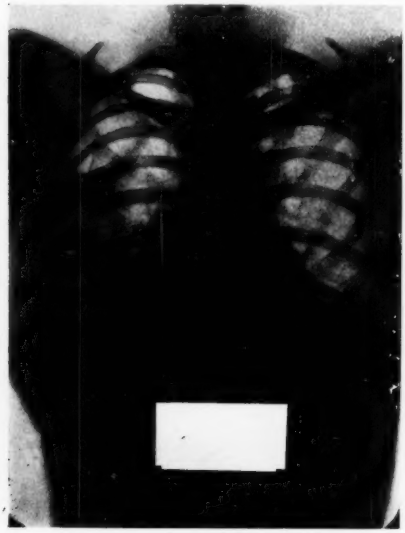

FIG 7.

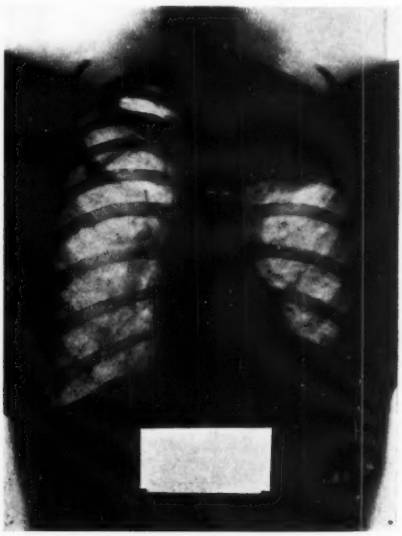

FIG. 8.

FIG. 7.—The artificial exudate is seen on the right side after glucose injection. The cavity in the left apex is considerably smaller (Case 4).

FIG. 8.—Shows the final result—viz., apical pneumothorax on the right side, apicolysis and phrenic paralysis on the left (Case 4).

to be a success, a sufficient breathing surface on the pneumothorax side must be obtained by lobe fixation *before* the intervention of diaphragmatic paralysis is performed.

Case 3.—A pneumothorax was induced owing to cavernous tuberculosis in the right upper lobe. The collapse was improved by cauterisation of adhesions. Thoracoscopy and stereoscopic radiographs showed that the middle and lower lobes were intact. There was already a spread of the disease from the cavity in the right lobe to the lower half of the left upper lobe in form of confluent infiltrations with honeycombed destruction. Therefore the middle and lower lobes were made adherent to the chest wall by means of the glucose method, and, as the result of this, the pneumothorax was confined to the right upper lobe. By this means sufficient breathing surface was obtained on the right side to allow the stiff-walled cavity in the upper lobe to be collapsed by frequent refills (at the commencement twice, and later once a week). In spite of this, the foci in the left lung showed no signs of improvement. Attempts to induce a double pneumothorax were unsuccessful. Improvement occurred only after phrenic paralysis of the left side. The jerky costal breathing which is to be observed with the combination of pneumothorax and phrenic paralysis on the contralateral side did not occur in this case, as sufficient breathing surface had been obtained by the artificial symphysis of the lower parts of the lung on the right side. The patient is now free from fever, there is no sputum, the cavity has disappeared, there is a definite regression of the foci on the left side, and the honeycombed destruction is no longer seen.

Apical Pneumothorax on One Side, Phrenic Paralysis and Apicolysis on the Contralateral Side.—The combination of pneumothorax on one side and apicolysis on the other has not such an unfavourable result as the combination of pneumothorax and diaphragmatic paralysis. The former only eliminates the function of the apex of the lung, and we know that the apex plays a less important part in the exchange of gases than the base. It is only when we are compelled to keep the lung well down that an additional strain is thrown on the contralateral side, and the danger exists of causing a spread of the disease in the focus underneath the plombage. Therefore, breathing surface should be obtained on the pneumothorax side before the intervention of apicolysis is performed.

Owing to the foreign body problem and the danger of the focus perforating into the paraffin, I only consider the intervention of apicolysis as an *ultimum refugium.**

* I reject the idea of paraffin plombage as a permanent condition. In pneumothorax the principle of a foreign body (air) also plays a rôle, although a less important one. However, we do not keep up a pneumothorax permanently, but when we consider that the lung is clinically healed we allow it to re-expand. Similarly, in my cases of apicolysis I only allow the paraffin plug to remain in position for so long as I consider it necessary for the focus to remain collapsed. It is an easy matter to remove the paraffin through

The more lung surface we can gain by the artificial fixation on the pneumothorax side, the less will be the strain thrown on the apicolysis side. The focus under the paraffin will have better chance of healing, and the danger of perforation is reduced.

As a supplement of the combination of pneumothorax on one side and diaphragmatic paralysis on the other, I will quote a case in connection with apicolysis.

CASE 4.—A case of severe bilateral phthisis with cavities in both apices. A pneumothorax was induced on the right side. The destructive process was favourably influenced by the collapse treatment, and the cavity in the apex of the contralateral side even became smaller. This improvement of the contralateral side was probably brought about by a weak mediastinum, whereby tension on the cavity was relieved.

A double pneumothorax failed owing to complete synechy. As the middle and lower lobes of the pneumothorax side were free from foci, an artificial fixation of these healthy parts of the lung to the chest wall was performed in order that the intervention of a phrenic extirpation might be effective. In a short time the patient was free from fever and sputum. The general condition improved considerably. X-ray examination showed that the cavity, previously the size of a fist, still remained, but was reduced to about the size of a grape. In order to avoid activity in this small cavity, the left apex was successfully collapsed by means of an apicolysis. For months the patient has been free from sputum and the general condition is excellent. As mentioned in the footnote, the plug will be removed at a later date, and the plombage bed covered by a small rib resection. The case which I have quoted would also have been suitable for a thoracoplasty. There is such a great amount of lung tissue eliminated by the combination of phrenic extirpation and apicolysis on one side that one might well have taken the responsibility for a thoracoplasty.

Lobe Pneumothorax and Thoracoplasty.

Up to the present time I have not had the opportunity of studying the combination of lobe pneumothorax and thoracoplasty. Owing to the extent of the intervention and the possibility of a pleural effusion on the pneumothorax side with its ill-effects, this form of double collapse treatment appears to me in general to be rather dangerous. However, this danger would appear to be reduced by the increasing extent of the region of the fixed lung.

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the original plombage incision. At the same time 3 to 6 cm. of the first four to five ribs are resected paravertebrally. By this means a superficial covering of the plombage bed is obtained. The remaining unnatural cavity fills with secretion, which later resorbs and organises. Through contraction the stumps of the ribs are drawn towards the remains of the plombage bed, and the lung re-expands to a certain extent. This re-expansion is not detrimental if the lung is really healed.

Cases of pleural thickening and contraction of the lung come under the same category of indications as the cases mentioned above. Finally, I would like to say a few words concerning collapse therapy on both sides.

The more complete the suppression of exchange of gas in one lung, the more hopeless is the outlook of collapse treatment on the other side, and therefore the gain of healthy lung surface on the contralateral side by artificial fixation in order to aid the respiratory function is of great importance.

The method of fixing the healthy parts of the lung to the chest wall requires further examination. It cannot be said for certain that 50 per cent. glucose is the best means to bring about fixation. Although up to the moment I have not experienced any unpleasant occurrences by my method, possible complications must be considered. It is possible, for example, that subpleural tubercle could be set free by the desquamation of the serosa endothelium, leading to a more or less severe tuberculous pleurisy. Further, one must consider the possibility of a perforation of the lung. On the other hand, if a perforation is so far advanced that it has penetrated to the endothelium of the visceral pleura, nothing can arrest its progress, and it matters little whether we have performed this intervention or not.

When one considers how often a pneumothorax is lost through a progressive pleural thickening, one might think that the artificial fixation may not stop at the desired spot, but could lead to a fixation of the pathological parts of the lung. Thoracoscopy has shown me, however, that progressive thickening of the pleura only occurs when either a fibrinous or sero-fibrinous pleurisy is present. The fibrin coagulum forms bridges and links between the pleural layers, and connective tissue and vessels grow in these links. Organisation and retraction of the fibrin masses causes the thickening.

My method brings about a temporary irritation of the healthy pleura. The inflammation of the visceral pleura over the diseased lobe is of no practical importance if we take care that there is enough air between the visceral and parietal pleura of the diseased lobe during the period of irritation.

Up to the present time I have limited this method to cases which, in my opinion, would have been lost without this method of lobar fixation. If the future shows fewer risks of complications, the indications can be extended.

The cases treated by this method require long, careful, clinical and radiological observation, and it is by no means suitable for ambulant cases.

THE TREATMENT OF TUBERCULOSIS BY HEAVY METALS, EXCLUDING GOLD, BUT WITH PARTICULAR REFERENCE TO THE USE OF CADMIUM

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The case of pulmonary tuberculosis which is unsuitable for any form of collapse therapy and does not respond satisfactorily to sanatorium treatment always presents a problem which excites the enthusiasm of the physician to attempt new methods, with the constant hope that the solution will be found which will benefit the individual and all subsequent patients. Amongst many other substances, the salts of heavy metals have attracted attention as therapeutic agents. Gold salts have been used most extensively, although their value is still strongly debated. Other heavy metals have been considered, but only used by occasional workers. The most important research on metal therapy is the work of L. E. Walbrum.[1] He has tabulated the value of a very large number of the salts of metals in the treatment of animals infected with various organisms. As regards tuberculosis, he divides metals into three groups according to their action on the tuberculous animal.

The only metals he considers to have any definite inhibitory effect on this lesion are barium, aluminium, cerium, selenium, cadmium, molybdium, ruthenium, and lanthium. A less marked effect was shown by platinum and eridium. Of all these metals, cadmium is the most convenient to use. A series of experiments was made with various salts of this metal and a number of patients treated. This work will be described later in this paper.

Iron.—On the argument that calcium conserves iron in metabolism and the two metals tend to increase together, Valy Menkin[2] has used repeated intravenous or intramuscular injections of ferric chloride in the treatment of four advanced cases. He gave 20 c.c. of a 0·0625 per cent. solution of a specially prepared solution of ferric chloride three times weekly. Apart from slight thickening of the veins, there were no untoward systemic deleterious effects, and he is of the opinion that " rigidly controlled and

cautiously carried out investigations on early or moderately early advanced cases of human pulmonary tuberculosis should be undertaken."

Copper.—Copper has been used by N. Mercador[3] in the form of colloidal copper morrhuate. He gave 5 c.c. intravenously or 10 c.c. intramuscularly on alternate days for periods of four months with two month intervals over a period of two years. He claims that the treatment produces a fall in pyrexia, clearing of the X-ray shadows due to tuberculosis, increase in weight, and elimination of tubercle bacilli from the sputum.

Copper has also been used by Stephani[4] in the form of mixtures of complex organic salts.

Copper benzoate, benzo-iodo-cuprol, and benzo-iodo-didyme were used suspended in oil.

The indications for this treatment are the same as those for gold salts, which it is able to replace completely.

One hundred and eight cases of pulmonary and surgical tuberculosis were treated: 72 per cent. improved, 21 per cent. were cured, and 19 per cent. remained stationary.

The doses given were 2 to 3 c.c. of the emulsion intramuscularly into the buttock every two or three days to a total of 200 to 600 c.c.

Stephani states that the copper compounds should be given alone in pulmonary cases, but the didymol will sometimes strengthen the action of the copper in the treatment of surgical tuberculosis.

Cadmium.—In 1927 N. Lund[5] treated thirty-eight patients with cadmium diuretin and claimed that twenty-four (63·3 per cent.) lost their tubercle bacilli. He describes a pleural and peritoneal reaction which simulated dry pleurisy after the injections, but reacted to digalin.

A further use of cadmium was made by Maigre and Reymier,[6] who treated six cases with an iodine-gold-cadmium compound with haxamethylenetetramine as a base; 100 c.c. of the solution contained 7 gr. iodine, 3 mg. cadmium, and 55 mg. gold. Five c.c. doses were given intramuscularly or intravenously twice a week up to 25 to 30 injections. They claimed reduction of fever and gain in weight in all six cases.

Cadmium Poisoning.—Cadmium has not been found in any living tissues except in the common scallop (*Pecten maximus*). In this species it appears to occur constantly.[7] In the human body there appears to be no cadmium.

A number of experiments were made by Prodau[8] on cats by introducing cadmium salts into their bodies by inhalation and ingestion. Using cadmium oxide fumes or dust, he found it produced increased rate of the respiratory rate, salivation, anorexia, vomiting and depression. Cadmium sulphide appeared to be very slowly absorbed, and symptoms were delayed for one or two days; then vomiting, dyspnœa, diarrhœa and occasionally salivation commenced.

The cadmium was excreted slowly through the kidneys and intestinal tract. There were no apparent changes in the blood picture. In very small amounts the cadmium powder produced scars in the lungs without subjective symptoms. Cadmium was found in the lungs, liver and kidneys shortly after exposure, and subsequently it seemed to be stored up in the liver, kidneys and lungs, and particularly in the bones. Permanent fibrotic changes were found in the lungs of all animals subjected to the experiments.

The Selection of a Suitable Cadmium Salt for Therapeutic Use.—Having reviewed the literature on the use of cadmium in the treatment of tuberculosis and given a brief account of the symptoms of cadmium poisoning, I will summarise the series of experiments which led me to select cadmium sulphide as the most suitable salt to use in treatment.

Experimental Work on Guinea-Pigs and Rabbits.

Experiment.	*Salt.*	*Doses Used.*	*Result.*	
			Local.	*General.*
No. 1.	Cadmium acetate.	0·5 or 1·0 c.c. of 1 per cent. solution subcutaneously.	Pain; temporary paralysis.	No general effect. Nothing abnormal. Post-mortem examination.
No. 2.	Cadmium glycine.	0·5 or 1·0 c.c. 25 per cent. solution weekly for 9 weeks subcutaneously.	Slight temporary pain.	Weight fell during injections, but increased later. P.M. normal.
No. 3.	Cadmium, potassium iodide, $Cd(KI)_2$.	1 c.c. of 5 per cent. solution subcutaneously.	All guinea-pigs died in 12 hours.	
No. 4.	Sodium, cadmium thioglycholate.	0·25 c.c. of 5 per cent. solution, 0·5 c.c. of 5 per cent. solution, 1·0 c.c. of 5 per cent. solution (subcutaneous).	None.	All guinea-pigs died in from 1 to 4 days.
No. 5.	Ditto.	0·5 c.c. of 1 per cent. solution, 0·5 c.c. of 2·5 per cent. solution (subcutaneous).	None.	All guinea-pigs died within 1 week.
No. 6.	Colloidal cadmium sulphide.	Weekly doses 0·25 c.c. of 0·2 per cent. for 13 weeks (subcutaneous).	None.	Guinea-pigs gained weight and showed no toxic symptoms.
No. 7.	Colloidal cadmium phosphate.	Ditto.	None.	Ditto.
No. 8.	Ditto.	0·5 c.c. of 0·2 per cent. weekly to 2 tuberculous guinea-pigs for 5 weeks.	None.	Animals died of tuberculosis in 5 weeks. Same time as controls.

No. 9.—A similar result was obtained using colloidal cadmium phosphate on infected rabbits.

No. 10.—Using colloidal cadmium sulphide, the treated rabbits lived 5 weeks longer than the controls, but it was inclined to produce small abscesses at the site of inoculation. Sections of the liver, lung and spleen of treated animals showed fewer tubercles than the slides of the controls.

Experiment.	*Salt.*	*Doses Used.*	*Result.*
No. 11.	Cadmium glycine.	0·5 c.c. of 2·5 per cent. solution twice weekly and 0·25 c.c. of 2·5 per cent. solution twice weekly subcutaneously to infected guinea-pigs.	Controls and treated animals killed at end of 5 weeks. Liver, lungs and spleen of treated animals showed slightly fewer tubercles.
No. 12.	Ditto.	0·04 grm. in 5 c.c. distilled water intravenously.	The rabbit died in 12 hours.
No. 13.	Sodium, cadmium tartrate	0·5 c.c. of 1 per cent. solution intravenously.	All rabbits suffered from dyspnœa and diarrhœa after each injection.
No. 14.	Sodium, cadmium thiosulphate.	1 c.c. of 1 per cent. solution intravenously.	Rabbit died in 12 hours.
No. 15.	Cadmium sulphide.	0·5 c.c. of 1 per cent. emulsion in oil subcutaneously twice weekly for 21 weeks.	All rabbits gained in weight. At P.M. lungs showed increased fibrosis. Livers enlarged and slightly fatty.

No. 16.—Twelve rabbits were given intravenously 20 mgms. of an emulsion of dead human tubercle bacilli. Four rabbits were treated with 0·5 c.c. cadmium sulphide, 1 per cent. suspension in oil, every third day. Four other rabbits were treated with 0·02 grm. per kg. weight of cadmium glycine subcutaneously every third day for 8 weeks. All rabbits were then given 1 c.c. bovine tuberculin (O.T.) intravenously. Two controls died immediately after this injection; 2 controls showed severe symptoms and were killed. The cadmium-sulphide-treated rabbits died after the injection of tuberculin. Two cadmium-glycine-treated rabbits died 8 hours after the injection and 2 recovered after 24 hours, but were killed later. At the post-mortem all the treated animals showed more fibrous tissue and fewer small tubercles in the lungs than the controls. This experiment was repeated, giving 0·5 c.c. of a 1 per cent. emulsion of cadmium sulphide in oil twice weekly for 10 weeks, as it was found that it was extremely difficult to obtain samples of cadmium glycine of constant composition and solubility. The second series gave similar results pathologically, although all the rabbits, both treated and controls, died immediately after the tuberculin injection.

No. 17.—The experiment was then repeated without giving the tuberculin, and at the end of 15 weeks all the treated rabbits had gained in weight and appeared healthy. At the post-mortem the usual increase in fibrous tissue was noted and a tendency for the treated animals to show fewer tubercles in the lungs.

No. 18.—Four rabbits infected with 0·02 mgm. human tubercle bacilli intravenously were treated for 14 weeks with bi-weekly subcutaneous injections of cadmium sulphide emulsion 1 per cent. Four rabbits were kept as controls. The controls all lost weight. Two of the treated animals lost weight and 2 remained stationary. At the autopsy all animals showed extensive miliary tubercles in lungs, but in 3 of the treated animals the lungs were clearer than the controls.

Conclusions of Experimental Work.

Cadmium glycine, cadmium sulphide, and colloidal cadmium sulphide were the only compounds non-toxic enough to be of therapeutic use.

Colloidal cadmium sulphide deserves further investigation.

Cadmium glycine should be useful if a product of constant solubility could be obtained.

Cadmium sulphide is non-toxic, painless, and appeared to stimulate fibrosis and exert an inhibitory action on the formation of tubercles.

Cadmium sulphide 1 per cent. emulsion in sterile olive oil was therefore selected for use in the treatment of patients.

Technique.

The emulsion is easily made in any laboratory. It is advisable to keep it in 50 c.c. sterile rubber-capped vaccine bottles with a few glass beads to prevent clumping, and to break up the sediment after the emulsion has been allowed to stand for a period. Before using it is necessary to stand the emulsion in warm water so as to facilitate filling the syringe.

The emulsion is given into the buttock in the usual manner. The patient need not rest or in any way change his normal routine after the injections.

The maximum dose I have given is 3 c.c. of a 1 per cent. emulsion, but I am of the opinion that much larger doses could be given quite safely and beneficially. There appear to be no contra-indications to the treatment, which can be given without reaction in the most advanced cases, even in those complicated by enteritis and laryngitis.

The emulsion is stable for long periods.

The results of treating twenty-seven cases have been tabulated as follows:

Case.	Age.	Lung Lesion.	Treatment.	Result of Treatment.					
				Temperature.	*Sputum.*	*T.B.*	*Weight.*	*X-rays.*	*General Result.*
1	30	*Right:* Infiltrated upper two-thirds, no cavitation. *Left:* Excavation upper lobe with infiltration. A.P.T. abandoned owing to adhesions and effusion	6 doses of 1 c.c. of 1 per cent. emulsion	Normal throughout	Increased from 2 ozs. to 8 ozs.	Remained positive	Increased 1 lb.	Excavation developed in right lung	Worse.
2	42	*Right:* Fibro-caseous lesion all lobes, with excavation in upper lobe. *Left:* Infiltration upper lobe	12 doses of 1 c.c. of 1 per cent. emulsion	Settled to normal	Decreased from 3 ozs. to 1 oz.	Remained positive	Gained 4 lbs.	Cavity became less and lesion became more fibrotic	Improved.
3	28	*Right:* Fibrotic lesion upper half with excavation. *Left:* Fibrotic lesion upper half	10 doses 1 c.c. and 8 doses 2 c.c. of 1 per cent. emulsion	Normal throughout	Scanty throughout	Negative (an A case)	Stationary	Remained much the same	Slightly improved Houghton's index improved from 270 to 284.
4	32	*Right:* Cases fibrotic lesion, with excavation upper half. *Left:* Cases fibrotic lesion, with excavation upper two-thirds	6 doses 1 c.c., 12 doses 2 c.c. of 1 per cent. emulsion	Normal throughout	Decreased from 1 oz. to nil	Positive to persistently negative	Gained 6 lbs.	Good deal of clearing in photograph	Clinically quiescent. Patient did heavy work and remained fit.
5	30	Fibroid lesion upper half both lungs. No excavation noted	8 doses 1 c.c., 14 doses 2 c.c. of 1 per cent. emulsion	Slightly febrile; became normal	Decreased from ½ oz. to nil	Positive to persistently negative	Gained 14 lbs.	No particular change	Clinically quiescent. Slight laryngeal lesion became arrested.

Case.	Age.	Lung Lesion.	Treatment.	Temperature.		Sputum.		T.B.		Weight.	X-rays.	General Result.
				Before.	After.	Before.	After.	Before.	After.			
6	35	*Right:* Fibroid infiltration. *Left:* Clear	8 doses 1 c.c. 1 per cent. emulsion	Slightly febrile	Normal	½ oz.	Trace	Neg.	Neg.	Increased 3 lbs. Decreased 2 lbs. previous to cadmium	No apparent change	On full work. Improved.
7	32	*Right:* Obliterating fibrosis due to old pleural effusion. Large cavity upper lobe. *Left:* Infiltration middle zone with cavity	25 injections 1 c.c. of 1 per cent. emulsion	Febrile	Slightly febrile	5 ozs.	7 ozs.	Pos.	Pos.	Lost 1 lb. in spite of 32 weeks hectic temperature	—	I.S.Q.
8	52	*Right:* Large apical cavity with dense infiltration of upper lobe. *Left:* Clear	14 injections of 1 c.c., 7 injections of 2 c.c., 3 injections of 3 c.c. of 1 per cent. emulsion	Febrile	Normal	1 oz.	½ oz.	Pos.	Neg. culture	Steady	Considerable increase of fibrosis and hardening of lesion	Improved. Pains in legs after injections.
9	18	*Right:* Massive infiltration middle and lower lobes, *Left:* Mottling middle third	7 injections 1 c.c., 20 injections 2 c.c. 1 per cent. emulsion	Febrile	Normal	2 ozs.	3 ozs.	Pos.	Pos.	Increased 5 lbs.	Lesions hardened	Laryngitis improved. G.C. improved.
10	41	Fibroid infiltration upper half both lungs	20 injections of 2 c.c. 1 per cent. emulsion	Slightly febrile	Normal	1 oz.	Trace	Pos.	Neg. culture	Gained 5 lbs.	No apparent change	On heavy work. Much improved. Clinically quiescent.

11	29	*Right:* Soft infiltration all lobes, with cavitation middle lobe. *Left:* The same	7 injections of 1 c.c., 18 injections of 2 c.c. 1 per cent. emulsion	Febrile	Normal	½ oz.	Trace	Pos.	Neg. culture	Gained 6 lbs.	Lesions hardened and became more discreet	Houghton's index improved from 74 to 155. Much improved.
12	48	*Right:* Fibroid infiltration middle zone. *Left:* Caseo-fibrotic lesion upper two-thirds, with excavation	9 injections 1 c.c., 14 injections 2 c.c. of 1 per cent. emulsion	Slightly febrile	Normal	1 oz.	Trace	Pos.	Neg. culture	Gained 16 lbs.	General hardening	Clinically quiescent.
13	44	Fibro-caseous lesion upper half both lungs	26 injections 2 c.c. 1 per cent. emulsion	Normal	Normal	½ oz.	Trace	Pos.	Pos.	Lost 2 lbs. before col., gained 5 lbs. on col.	No apparent change	Improved.
14	32	Fibro-caseous infiltration left lung. Early infiltration right upper lobe	8 injections of 1 c.c., 12 injections of 2 c.c., 10 injections of 3 c.c. 1 per cent. emulsion	Slightly febrile	Normal	1 oz.	½ oz.	Pos.	Neg.	Gained 6 lbs. on col.	No apparent change	Improved.
15	31	Extensive disease right side and upper half left	26 injections of 2 c.c. 1 per cent. emulsion	Slightly febrile	Normal	2 ozs.	½ oz.	Pos.	Pos.	Gained 14 lbs.	Appearance hardened.	Improved.
16	31	*Right:* Artificial pneumo-thorax. *Left:* Infiltrated middle zone	8 injections of 1 c.c., 38 injections of 2 c.c. 1 per cent. emulsion	Febrile	Normal	2 ozs.	2 ozs.	Pos.	Pos.	Stationary enteritis developed	No apparent change	Houghton index dropped from 113 to 85. Worse.
17	21	Extensive fibroid lesion all lobes	7 injections of 1 c.c., 21 injections of 2 c.c. 1 per cent. emulsion	Normal	Normal	1 oz.	1 oz.	Pos.	Pos.	Gained 9 lbs. on leaving work	No apparent change	Houghton index increased from 200 to 252. Improved.

Case.	Age.	Lung Lesion.	Treatment.	Temperature.		Sputum.		T.B.		Weight.	X-rays.	General Result.
				Before.	After.	Before.	After.	Before.	After.			
18	45	Fibro-caseous infiltration upper half of both lungs, with cavitation	8 injections of 1 c.c., 6 injections of 2 c.c., 10 injections of 3 c.c.	Normal	Normal	1 oz.	Trace	Pos.	Neg.	Gained 5 lbs.	—	Improved.
19	38	*Right:* Clear. *Left:* Early infiltration upper third	Left temp. phrenic. 3 injections 1 c.c., 2 injections 2 c.c., 12 injections 3 c.c. 1 per cent. emulsion	Normal	Normal	½ oz.	Nil	Pos.	Neg. culture	Stationary	Apparent clearing of lesion	Improved.
20	21	Early infiltration upper third both lungs. No cavitation	5 injections of 1 c.c., 35 injections of 2 c.c. 1 per cent. emulsion	Normal	Normal	1 oz.	Trace	Occas. Pos.	Neg.	Gained 8 lbs.	—	Houghton index improved from 84 to 134. Improved.
21	33	*Right:* Extensive fine infiltration all lobes. *Left:* Extensive fibro-caseous infiltration all lobes with multiple cavities	9 injections of 1 c.c. 1 per cent. emulsion	Hectic	Hectic	2 ozs.	4 ozs.	Pos.	Pos.	Lost 5 lbs.	—	Worse.
22	22	*Right:* Fibro-caseous infiltration upper half with two cavities. *Left:* Infiltration upper one-third	Right temp. phrenic. 15 injections of 1 c.c., 3 injections of 2 c.c. 1 per cent. emulsion	Hectic	Normal	1 oz.	1 oz.	Pos.	Pos.	Lost 14 lbs.	Disease progressing	Worse.

23	24	*Right :* Clear. *Left :* Infiltration upper third	4 injections of 1 c.c., 10 injections of 2 c.c., 7 injections of 3 c.c. 1 per cent. emulsion	Normal	Normal	½ oz.	Trace	Pos.	Neg.	Gained 4 lbs. on hard work	Slight clearing	Improved.
24	25	*Right :* Infiltrated upper half with excavation. *Left :* Early infiltration	2 injections of 1 c.c., 8 injections of 2 c.c., 12 injections of 3 c.c. 1 per cent. emulsion	Normal	Normal	½ oz.	Trace	Pos.	Neg. culture	Gained 7 lbs.	—	Improved.
25	43	*Right :* Fibrous infiltration all lobes. *Left :* Fibro-caseous infiltration upper two-thirds with large cavity	16 injections 2 c.c.	Slightly febrile	Normal	1½ ozs.	½ oz.	Pos.	Pos.	Gained 6½ lbs.	—	Improved.
26	34	Fibrous infiltration upper half both lungs	3 injections 1 c.c., 14 injections 2 c.c.	Slightly febrile	Slightly febrile	1 oz.	1 oz.	Pos.	Pos.	Gained 5 lbs.	—	Stationary.
27	28	*Right :* Infiltration upper half with cavitation. *Left :* Early infiltration upper lobe	20 injections of 2 c.c.	Normal	Normal	½ oz.	Nil	Pos.	Neg. culture	Gained 19 lbs.	Considerable clearing	Clinically quiescent.
28	32	Infiltration upper half of both lungs. No cavitation	15 injections of 2 c.c.	Slightly febrile	Normal	½ oz.	Trace	Pos.	Pos.	Gained 15 lbs.	Considerable clearing	Improved.

Summary of the Immediate Results.

Number of cases treated	27
Clinically quiescent	4
Improved	17
Stationary	2
Worse	4
Number in which tubercle bacilli disappeared from the sputum on repeated examination and culture (two cases never gave positive sputum)	13 out of 25

Twenty cases gained a considerable amount of weight when put on cadmium treatment.

Eleven cases showed improvement in the radiograph, particularly in the hardening of the shadows and clearing of woolly and fluffy shadows.

Nineteen cases showed diminution in the amount of sputum.

One case complained of pains in the limbs after the injections and one case had periods of depression during the treatment. These were the only constitutional disturbances.

Conclusions.

Heavy metals have a definite place in the pharmacopœia of the phthisiologist, and although gold has had by far the most attention, other metals can give results which are as good, and by further trial may be better than those so far obtained with gold.

Cadmium in the form of the sulphide or glycine is worthy of consideration. From the results obtained in the very small number of cases little definite can be concluded, except that the treatment is harmless and appears to give immediate beneficial results. They appear quite comparable with those obtained with gold salts, and in view of the very small cost of cadmium compared with that of gold, further work on the subject is desirable.

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AIR EMBOLISM AND SPONTANEOUS PNEUMOTHORAX COMPLICATING ARTIFICIAL PNEUMOTHORAX*

By J. A. MYERS, IDA LEVINE and ELIZABETH A. LEGGETT

Two serious complications which occasionally result from common surgical procedures used in the diagnosis and treatment of diseases of the lungs and pleura are air embolism and accidental pneumothorax.

Accidentally Brandes[1] demonstrated the mechanism of air embolism. In 1912 he reported a case in which he tried to outline the extent of an empyema cavity with bismuth paste injected through a sinus. The patient had a spasm and there was deviation of the eyes. Death occurred in twenty hours. At autopsy bismuth was found in the smallest bloodvessels of the cerebral cortex on both sides. Serial sections made from the walls of the sinus revealed bismuth as far as the pulmonary capillaries and veins. Other capillaries of the systemic system also contained bismuth. The rubber catheter used to inject the bismuth had produced mechanical injury so as to open some of the very small veins, thus allowing the bismuth to enter.

Other workers have observed among their patients such symptoms as eye changes, consisting of dilatation of the pupils, failure of the pupils to react to light, deviation of the eyes, and in some cases blindness; headaches, dizziness, and paresthesias also occurred; tonic and clonic convulsions were observed, and even if the patient recovered, paralysis frequently would persist for several days; aphasia and loss of consciousness were observed; and in a few cases there was vomiting. Some patients have died soon after the onset of the symptoms. Less severe symptoms, such as irregularity in respiration and abnormalities in the action of the heart, have been reported. The similarity of symptoms and outcome between these cases and Brandes' case suggested the question as to whether they were not due to air gaining admission to the small bloodvessels of the lung. Leon-Kindberg,[2] Olmer and Turries,[3] Vandriessche,[4] Cocke,[5] McKnight, Gammons, and Knowles[6] have reported cases in which they attributed the symptoms produced to pleural shock. Vandriessche even goes so far as to state that " the theory of gas embolism should be abandoned."

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Laurenti[7] describes three types of reactions accompanying puncture of the pleura. They are syncopal, convulsive, and hemiplegic. The various modifications of these reactions are of autonomic or sympathetic nervous origin. He believes that those more commonly seen are referable to the autonomic system. However, he is convinced that some of the reactions are due to gas embolism, and many of the transitory disturbances, such as mild paralytic symptoms and paresthesias, are due to cerebral ischemia from vasospasm.

Brauer[8] in 1913 was convinced that all the serious accidents are due to air embolism. Since his report this view has gained favour with the majority of physicians who have seen such accidents. However, others are convinced that the symptoms, particularly in the mild cases, are due to irritation of the pleura producing " pleural shock " or " pleural eclampsia." In the fatal case it is often difficult at autopsy to determine the cause of death.

Cordier (cited by Riviere[9]) induced pleural shock by chemical irritation and prevented it by anæsthesia or by ligation of the carotids—*i.e.*, pressure on the vagus. Croizier,[10] repeating these experiments, but controlling them by manometric readings, induced the convulsions, crises, and death described by Cordier only when he injected the irritant into the lung tissue itself. Administration of iodine and other irritants into the pleural cavity occasionally caused immediate shock, but did not bring about convulsions or death. He is of the opinion that embolism is the cause of this symptom complex, and in support of it cites Cordier's statement that it could be prevented by ligation of the carotid arteries.

Rukstinat and LeCount[11] reviewed the literature concerning air embolism and reported the results of their experimental work. By means of a tracheal cannula, air under pressure was forced into the lungs. It entered the pulmonary veins when the pressure was above 30 mm. At about 30 mm. pressure convulsions were invariable, being first tonic then clonic. In all the animals some pneumothorax was present. Air was also present in the aorta, in the carotid, brachial, and iliac arteries, and frothy blood was present in all the heart chambers. There was always air in the coronary arteries.

In animals, when one mechanically irritates the normal pleura, there is no constant specific action on the central nervous system. With stimulation of the parietal pleura slight protective movements are observed, or in some animals local clonic contractions are produced on the same side. But there are no visible results on stimulation of the pulmonary or mediastinal pleura. Some believe that sensory stimuli are carried over the fibres of the vagus nerve, but in old empyema cases the changes in the pleura, particularly the extensive fibrous tissue, may completely block the stimuli.

This leaves only the sympathetic nerves with their sensitive element running with the bloodvessels, and these nerves have no influence on protective movements. In empyema cavity walls, bloodvessels are seen suspended between strands of fibrous tissue. Schlaepfer[12] has pointed out that when such vessels are injured their walls cannot contract well on account of the inelastic attachments. Therefore, the opening may persist and air or gas be permitted to enter from a needle or an empyema cavity, since there is already a negative pressure in the veins. When injury is done to the normal lung parenchyma, the bloodvessels are capable of reducing their size and their lumina so quickly that little or no air is admitted. But if injury is done to diseased lung tissue, the vessels having lost their ability to contract, air may enter from alveoli or from a needle such as that used in artificial pneumothorax.

Wever[13] has shown experimentally that when air to the amount of several cubic centimetres is injected into the carotid artery of animals retinal changes occur. He has seen large numbers of bubbles and short columns of air in the retinal arteries. These give the retina a bright silvery effect. In a very short time these arteries become completely filled with air and the optic disc appears almost white. Throughout the fundus in the region of the macula many bright stripes are seen to form a network. In two or three minutes the air begins to disappear, and in five minutes the retina appears normal. Similar observations have been made by ophthalmologic examination of the human eye following accidents.

The mechanism of gas embolism is that of the introduction of air into the systemic arteries by way of the left heart from the pulmonary veins. Air may enter these veins from three sources: the alveolar air, the intrapleural air, or the air in the pneumothorax apparatus. This may be brought about by puncturing a vein or indirectly through injury of the visceral pleura or lung parenchyma. If the needle pierces the lung, and because of fibrosis, adhesions, or other pathology, the tissue is unable to contract and seal the opening at once, air from the alveoli, the pleural space, or the needle may be aspirated into the transfixed and injured pulmonary vessels.

Sorgo[14] believes that sudden death is caused by failure of the right heart or by obstructing the coronary arteries or vital cerebral centres. He is of the opinion that small emboli may occur in various regions without serious or permanent damage.

Riviere,[9] Bishop,[15] and Siebert[16] have given excellent discussions of the mechanism of air embolism. The aspiration of air into the veins is especially apt to occur with high intrapleural pressure. The intravenous pressure is so low that air may be aspirated even if the intrapleural air pressure is low. Piper[17] found the pressures in the jugular veins of cats and dogs vary from – 12

cm. of water on expiration to – 14 cm. during quiet inspiration and to – 19 cm. on deep inspiration. Riviere considers that the suction power in man might amount to – 7 or – 8 cm. of water. Hofuendahl[18] advises the use of an artificial pneumothorax apparatus in which the pressure in the connecting tubing and needle is less than those anticipated in the pleural cavity. The aspiration of small amounts of air into the systemic veins is usually not of great importance, the emboli being trapped in the pulmonary circulation. The occurrence of air embolism has been markedly reduced since the injections have been controlled by manometric observations.

Another argument in favour of the symptoms being due to gas embolism is that air has been found in the vessels of the brain of some cases at post-mortem. Moreover, in other cases where air could not be demonstrated in these vessels definite injury to certain regions of the brain was found to exist. Siebert[16] had two fatal cases in which there were tissue changes in the brain, but no actual air bubbles could be seen. Bishop reported one fatal case in which the right vessels to the brain contained air bubbles. Saugman[19, 20] reported two cases with fatal termination. In one, air emboli were found in the subarachnoid veins, in the veins of the base of the brain, and in one cerebral artery. In the second, the basal vessels to the fossa of Sylvius were air filled. Hochstetter[21] called attention to a case in which petechial hæmorrhage was seen in the brain at post-mortem. Pollak[22] reported one case in which air was found in the coronary arteries and heart chamber. The head was not examined.

In 1922 Schlaepfer[12] made a very extensive study of air embolism following various diagnostic or therapeutic procedures in diseases of the pleura and the lung. A part of his conclusions are as follows: "The sudden release of a great amount of fluid in emaciated people with a neurotic constitution (neurasthenia, epilepsy) may cause a similar clinical picture. Because of its slightness and short duration, with lack of localised cerebral symptoms and serious sequelæ, this condition is considered to be due to shock. Chronic myocardial lesions or an insufficiency of the adrenals may cause sudden death during the performance of one of these operations on the chest wall. A similar clinical picture is seen in embolism of the pulmonary artery. Death, accompanied by these signs, may occur also in a few cases of emboli in the brain following thrombosis of pulmonary veins. The autopsy will clear up the diagnosis. It was demonstrated by experiments that we do not have a pleural reflex, even in the normal pleura, which would explain these complications. They are proved by the accidents in pneumothorax therapy to be due to air emboli."

In some cases following accident, albumin and casts occur in the urine. This finding is believed to be due to air temporarily blocking the renal

system. Also one must not overlook the possibility of thrombi accounting for the symptoms in some cases. We know that in chronic inflammatory conditions of the lung some of the small vessels may be thrombosed and manipulation may be sufficient to release thrombi.

The question of time of occurrence of symptoms is of considerable significance. Ranzi and Albrecht[23] cite three cases of sudden unconsciousness occurring during operations involving the pleura. Two of these patients died. They believe that each of the three had air embolus. Hornung[24] also reports a case of air embolus occurring while a rib resection was being performed on a patient with lung abscess. Reyer and Kohl[25] called attention to ten patients on artificial pneumothorax treatment who developed air embolism, seven of whom were in the sitting position or attempting to arise from the table when the symptoms appeared. In 40 per cent. of their patients symptoms were observed without the actual introduction of air. In four nausea was the first symptom; six became totally unconscious. It is significant that nine of the ten patients showed evidence of adhesions on X-ray examination. They believe that when the symptoms appear before air is introduced the air is aspirated from the needle and connecting tube from the pleural cavity, or from air spaces in the lung. Therefore they occur during surgical operations and during the passing of a needle through the parietal pleura. In cases of artificial pneumothorax they may result from air entering a bloodvessel directly from the needle, but no doubt they more frequently occur when adhesions give way, allowing air to enter vessels. Therefore, even at any time between treatments air embolism is possible.

How often do serious complications occur? Zesas[26] collected from the literature fifty-four cases with serious symptoms accompanying or following manipulation of the pleura. In ten thousand punctures Forlanini[27, 28] had twelve accidents, five of which were serious enough that pneumothorax treatment had to be abandoned. Saugman,[19, 20] in the period from December, 1906, to March, 1921, treated five hundred and thirty patients with pneumothorax, making eleven thousand punctures. Two patients died suddenly, presumably of gas embolism, although the diagnosis was not confirmed at post-mortem. Sachs[29] collected from the literature 1,058 cases treated by American authors, in which series three cases of air embolism and twenty-six cases of pleural shock occurred. Sachs reported seventy-four cases of shock in seven hundred injections in his own experience. Bishop,[15] summarising the literature, considered the incidence of air embolism to be one in one thousand or one in fifteen hundred refills. Matson *et al.*[30] report four cases of embolism (two were fatal) in twelve thousand inflations. Cooper and Stallings[31] had ten cases in four hundred and eighteen pneumothoraces. Kohlhaas[32] reports two cases in three thousand fillings, repre-

senting three hundred patients treated. Bruns[33] had sixteen cases in twelve thousand punctures. Siebert[16] had two cases in ten years' experience. Swezey and Schonbar[34] during ten years' experience, in which two hundred and seven cases were treated, had three with gas embolism and pleural shock, one of which terminated fatally. Andrews[35] in a series of 8,528 needle punctures, in 8,085 of which air was introduced, saw six cases with alarming symptoms. One was a reaction to novocain, two were syncope, one was extreme nervousness, two were pleural shock or air embolism, one of the two being fatal. Borock and Wildre[36] observed air embolism twelve times in a total of 13,935 refills. They believe that such emboli originate when the needle enters a pulmonary vessel, particularly at the time of the first treatment, or when the needle enters an adhesion at the time of a refill, or when air enters a vessel in the torn band of adhesions. In twelve thousand treatments Maendl[37] had no serious or unfortunate accident occur, while Croizier[10] had no accident in ten thousand pneumothorax treatments.

Freund[38] reported two cases of air embolism who recovered. Sardo[39] cited a case in which a pneumothorax refill was followed by a flaccid monoplegia of the right arm. He attributed it to air embolism. Besançon, Azorelay, and Chaubaud[40] reported ten cases of air embolism and pleural shock. Rekonomopoulo and Papanikalau[41] had two cases in which they believed the symptoms might be due to either pleural reflex or air embolism. Olmer and Turries[3] reported one case of pleural shock which occurred as the needle pierced the pleura.

Bruns[33] called attention to sixteen cases of air embolism with seven deaths. Cooper and Stallings[31] reported ten cases of air embolism with three deaths. Siebert[16] had three embolic accidents with two deaths. Matson *et al.*[30] cited four cases of air embolism with two deaths. Pollak[22] had three cases with one death. Bishop[15] reported ten cases of air embolism with one death in his own practice, and stated that the literature gave a mortality of 30 to 50 per cent. In the fifty-four cases Zesas[26] collected from the literature twenty-one were fatal. Two of Saugman's[19, 20] cases died. In 1913 Sundberg (cited by Sercer and Peicic[42]) reported three deaths, and one year later Sillig,[42] Begtrup-Hansen[42] and Jessen[43] each reported a death. In 1920 Link[42] and Wolff-Eisner[42] each saw one fatal case, and the same year Siebert[16] had two fatal cases. In 1921 Mory[44] reported a fatal case, and the next year Kowitz[45] had two deaths. In 1923 Stivelman,[46] Warner and DuBray[47] each reported a death. A year later Hochstetter[21] had a case which proved to be fatal. In 1925 Leon-Kindberg[2] and Bishop[15] each reported a case, and the same year Besançon[40] reported four deaths. In 1926 Lindblom[48] recorded a fatal case. Lauret and Fontan have had one death, and Giese and Conway also have reported one death.

During the operation the patient should be observed closely, and at any indication of trouble, such as unusual restlessness, deviation of the eyes, irregularity of respiration or pulse, muscular twitchings or pallor, the needle should be withdrawn immediately. Should embolism occur, the treatment usually advised is to keep the patient absolutely quiet in a reclining position with the head lowered. The heart action should be strengthened by the use of adrenalin or strychnin. In cases of coronary air embolism or cardiac failure adrenalin may be injected directly into the heart. If the patient is restless, morphine in small doses is helpful. Friction may be used in the region of the heart. Some authors who believe the symptoms are due to pleural shock advocate the use of artificial respiration. Others are opposed to this procedure, believing that more air may be pumped into the injured and open walls of the bloodvessels.

In the prevention of both pleural shock and air embolism careful technic is of extreme importance. The site of puncture should be as far as possible from the area of disease. All possible injury to the lung and pleura should be avoided. Satisfactory manometric readings must be obtained before any air is given, and the patient warned to avoid deep breathing and coughing during the treatment. Sudden changes in intrapleural pressure should be avoided.

Forlanini[27, 28] in 1912 stated that with careful technic air embolism should be of historical interest only. Many physicians, particularly those who believe in pleural shock, place great emphasis upon the mental attitude of the patient. The pre-operative administration of morphine or some other sedative is frequently advocated. Many believe that accidents may be avoided by the use of novocain in every case, but Stivelman,[46] after making a study of the literature, pointed out that accidents have occurred more often with the use of local anæsthesia than without it. Obviously, careful manometric readings will prevent the introduction of air into the vessels, but we are strongly of the opinion that this is not the common cause of gas embolus, but that the tearing of adhesions is the usual cause; therefore, we believe that the only way to prevent the occasional accident is to institute artificial pneumothorax while the disease is minimal and before adhesions have formed in any considerable number of cases. In a recent paper[49] we called attention to the fact that in fifty-two of our cases who had artificial pneumothorax instituted when the disease was minimal, only three had adhesions which interfered with satisfactory collapse of the lung. In none of these cases has gas embolus occurred. Moreover, among patients treated by ambulatory artificial pneumothorax[50, 51] from the beginning there apparently is no more likelihood of this complication developing than among those treated by artificial pneumothorax and strict bed rest.

In performing pneumothorax since 1920 with a present group of approximately one hundred and fifty patients we have had two slight reactions and three more serious ones. The first was a man who had been on artificial pneumothorax treatment for more than a year. The pleural cavity was becoming obliterated through the formation of adhesions, so much so that only a small amount of air (50 to 100 c.c.) could be introduced at each refill. Immediately following a treatment he complained of considerable dizziness and some nausea. The pupils became somewhat dilated, but he was not unconscious, nor were there any evidences of paralysis. Since it was impossible to introduce very much air at a time and there was some question of the benefit he was deriving from the treatments, it was decided to allow the lung to completely re-expand.

The second patient was a young woman who had been on pneumothorax treatment for more than a year and had extensive adhesions. One day after her treatment, while still lying on the table, she complained of considerable dizziness and a peculiar sensation which she could not explain. A little later she became extremely pale, her heart was slightly irregular, but the symptoms disappeared in a few minutes. She had derived so much benefit from artificial pneumothorax treatment that it was continued.

Of the four patients with more severe reactions, one was a woman who had developed obliterative adhesions. On one occasion after the needle was introduced and 10 to 15 c.c. of air allowed to flow through the needle she almost instantly began to scratch her upper lip and in another moment was unconscious, and remained so for about four hours. Her pupils were dilated and she developed marked rigidity of the right upper and lower extremities. Her pulse and respirations became irregular. She was extremely pale. Soon after the attack started her head was lowered, and adrenalin was administered subcutaneously. She stated that the only sensation before losing consciousness was that of a fly on her upper lip. At the end of four hours she was transferred to a hospital, where she remained one week. She developed a paralysis of the right upper and lower extremities which lasted approximately three days, but she made a complete recovery and decided to have her artificial pneumothorax treatment before leaving the hospital.

The second serious case is that of a young woman receiving artificial pneumothorax on the left side. There were numerous adhesions, so that only small amounts of gas could be administered. One day, just as the treatment was being started, and when only a small amount of air had been introduced, she suddenly lost consciousness and cried out. The left side of her tongue became markedly blanched, a symptom to which Liebermeister[52] and Michalowski[53] have called attention. She was very pale, frothed at

the mouth, and it required considerable effort to keep her from biting her tongue. Her heart became extremely irregular and respiration almost ceased. Adrenalin was administered and in about two hours the symptoms began to disappear, yet she was only semi-conscious. It was three days before all symptoms had disappeared. She has no memory whatsoever of what happened at any time during the first four hours after the attack. She continued on artificial pneumothorax for some time, but the collapse was later completely lost through obliterative adhesions.

The only fatal case we have seen was that of a woman of twenty-nine years, who had artificial pneumothorax instituted about four years before for extensive pulmonary tuberculosis. She had numerous adhesions, but the pneumothorax had brought her disease well under control. Her general condition was excellent, and she had become quite active physically. Following a treatment, when the usual amount of air was introduced with good manometer readings and no unusual experience, she raised herself up on the pneumothorax table, but instantly fell back in an unconscious state and died in a few minutes. The various procedures which have been recommended, such as the administration of adrenalin into the heart, were carried out, but none was of any avail. This occurred in 1929, and in the seven years that have since elapsed we have not seen even the slightest symptom of gas embolus in any patient.

Accidental or spontaneous pneumothorax sometimes complicates artificial pneumothorax. This may occur on either side, but the incidence on the artificial pneumothorax side is greater. Figures on the frequency of this complication are indefinite, since many physicians do not consider lung fistulæ under the same heading as " spontaneous " pneumothorax. Moreover, some of the cases remain undiagnosed until effusion or empyema forms, whereupon they are classed under those headings. However, the incidence of this complication is given by the following authors:

Forlanini[27, 28]	6 per cent.
Dumarest and Brette[54]	4 ,,
Burrell[55]	2·3 ,,
Hutchinson and Blair[56]	5·8 ,,
Cooper and Stallings[31]	8 ,,
Swezey and Schonbar[34]	3·8 ,,
Matson *et al.*[30]	3·3 ,,
Bronfin[57]	14 ,,

This condition is usually due to direct injury to the lung or visceral pleura by the pneumothorax needle, spontaneous rupture through a weakened subpleural area, or tearing of an adhesion or empyema burrowing through the visceral pleura. The accident may occur immediately after chest puncture or be delayed for some time after the filling. The fistula may be of

the open, valvular, or closed types; and the prognosis to great extent varies accordingly.

The closed type, which has the best prognosis, is usually due to direct puncture of healthy lung tissue, although it is conceivable that a few might be due to spontaneous rupture of the visceral pleura. Practically all of them come under control without complications or treatment within a few days or weeks.

The valvular and open types of fistula constitute the serious cases, and are probably most frequently caused by rupture of adhesions. Heise and Krause[58] reported a case who, the day after artificial pneumothorax was performed, after raising himself in bed, developed sharp pain on the pneumothorax side. He died fourteen hours later. The post-mortem examination showed that no injury had been caused by the needle, but there was a tear in the pleura over a small cavity near an adhesion. The thoracic cavity contained about two litres of clotted blood. Roch and Saloz[59] reported a case of a woman who, following the twelfth artificial pneumothorax treatment, developed empyema. They were of the opinion that this was due to a tear in the lung resulting in a fistula. The resulting pyopneumothorax healed in four months and the fistula closed. Bard[60] believes that tearing of adhesions during the course of artificial pneumothorax therapy is likely to cause fistulæ, and that this is a frequent cause of pleural effusions. Lux[61] is of the opinion that lung perforation by tearing adhesions is much more frequent than commonly believed and often is not serious. He believes that frequently it is the cause of effusions which appear in the pneumothorax cavity. Cicconardi[62] reported four cases in each of whom the symptoms were of short duration. He believes that spontaneous pneumothorax results either from a tear of the pulmonary parenchyma or injury from the needle.

Spontaneous rupture of a cavity or caseous area is perhaps next in frequency. Maendl[37] reported a case who developed stabbing pains fourteen days after the administration of pneumothorax. His temperature increased to 104°, marked dyspnœa was present, and his pulse was rapid. The post-mortem showed a small opening leading from a pulmonary cavity into the pleural cavity. Jessen[43] believes that spontaneous pneumothorax usually is brought about through rupture of a cavity. However, he reports a case with this complication, but at post-mortem no cavity could be found. Instead there were two tuberculous nodules which extended through the visceral pleura.

Direct injury to a cavity or caseous focus by the pneumothorax needle is another cause of accidental pneumothorax. This is much more serious than injuring healthy tissue, not only because diseased lung has lost much of its resiliency and recuperative powers, but also because of the danger of

spreading the infection from this source. Tobe and Terrasse[63] reported two cases of lung perforation occurring after the first artificial pneumothorax treatment. The fistula healed spontaneously in each case. Wallgren[64] had two cases of spontaneous pneumothorax complicating artificial pneumothorax. In one, this complication occurred twice. The first time he believed it was due to the needle puncture, but second to the giving way of a linear adhesion. There was no serious result either time. In his second case the spontaneous pneumothorax did not become evident until eight hours after the treatment. He believes that spontaneous pneumothorax can occur from progress of the destructive process, from trauma, or from traction on a weak part of the lung, or from perforation into the lung tissue of a pleural empyema complicating the artificial pneumothorax. Herve and Roussel[65] believe that pyopneumothorax in artificial pneumothorax cases is generally due to perforation of the lung.

Rupture of a pyopneumothorax into a bronchus occasionally results in spontaneous pneumothorax. Among Duboff's[66] twenty cases of tuberculous empyema, four ruptured into bronchi. Riviere[9] had three such cases. But on the whole this cause is not so important numerically as the three preceding.

Spontaneous or accidental pneumothorax is more frequent in far advanced disease, both because this presents the most pathology, and so the most chance for rupture, and also because it presents the bulk of cases treated. Moreover, it tends to occur early in the treatment. Of the eighteen cases Rist[67] discusses, twelve occurred within five months of treatment, five after one year of treatment, and only one after three years.

In bilateral therapeutic pneumothorax Chaubaud[68] observed acute perforations in nine out of thirty-four cases. This is a higher percentage than he has observed in unilateral pneumothorax. He believes that the risk of tearing the pulmonary tissue is much greater in bilateral cases.

The onset and symptoms of accidental pneumothorax vary from sudden excruciating chest pain associated with severe cardio-respiratory embarrassment and shock to absence of symptoms. Diagnosis can be made definitely by the manometer. With an open fistula the readings will be approximately those of the intrapulmonary pressure; with a valvular fistula positive pressures recur in spite of the aspiration of air.

The complications are threefold: occurrence of fluid (infected or not), persistence of the bronchial fistula, and finally aggravation of the disease. Wilson[69] is of the opinion that some effusion develops in at least one-half of the cases of true spontaneous pneumothorax, and at least half of them are purulent; the figures for accidental pneumothorax are higher.

The prognosis varies with the type of fistula, the complications, etc. Burnand[70] reports nine cases of spontaneous pneumothorax accompanying

artificial pneumothorax, three of whom died the first month and four others in three to six months from empyema. Bronfin[57] had a mortality of 25 per cent., Sauerbruch[71] found the mortality to be 25 per cent.; yet he states that of fifty-seven cases of cavity rupture, forty-three were dead within a week, and the total mortality was 86 per cent.

Treatment is symptomatic, with reduction of intrapleural pressure whereever indicated. The empyema should be treated by aspiration in so far as possible, and if this does not control the infection, radical surgery is indicated. In cases of permanent bronchial fistulas, Straub,[72] Alexander,[73] and others find that thoracoplasty offers the only hope for recovery. Oleothorax may be justified in small fistulas both as a curative agent and as a means of improving the patient's condition for operation; it is inadvisable in large fistulas, since it may flood the contralateral lung. Oleothorax is also of some value in combating the empyema.

We have seen two cases which we believe were due to accidental or spontaneous pneumothorax, but our diagnosis was based entirely upon the symptoms given by friends and relatives. The first was that of a woman of thirty-seven years. Artificial pneumothorax had been instituted several years before, and she had been restored to normal living conditions. At the time artificial pneumothorax was instituted her disease was far advanced and large cavities were present. She had numerous adhesions which were slowly obliterating the pneumothorax space. After being discharged from the sanatorium she had led a normal life for several years. After an uneventful treatment she drove her car home. She experienced some shortness of breath several hours later. This was not considered significant by her associates, who did not report the condition until late in the evening, when the dyspnœa had become quite severe. She died before we were able to reach her home.

The second case was that of a woman of twenty-five years, who had extensive disease involving the left lung, diagnosed nine years before. She had done well on artificial pneumothorax treatment, although numerous adhesions were present; in fact, she had been restored to quite normal living conditions. After receiving her treatment as usual one afternoon the fluoroscopic examination revealed no change from the usual findings. When she arrived home after several hours she was very dyspnœic, but entirely conscious. She died about fifteen minutes later. In neither of these cases were post-mortem examinations made, but the symptoms described were those of spontaneous or accidental pneumothorax rather than those of embolus. Among fifty-two cases with artificial pneumothorax instituted while tuberculosis was in the minimal stage we have seen only one case of pulmonary fistula. This occurred in a patient who had been on treatment

for several years and did not change the status of the collapsed lung. In this group we have seen no case of accidental pneumothorax.

In the prevention of accidental pneumothorax on the artificial pneumothorax side, as well as the complications which follow this accident, many suggestions have been offered. In our opinion, just as in the case of gas embolus, the tearing of adhesions is the common cause of this accident. Therefore, patients who have artificial pneumothorax instituted before adhesions make their appearance are less likely to suffer from accidental pneumothorax. Since the rupture of a pulmonary cavity into the artificial pneumothorax space is a common cause of accidental pneumothorax, we are of the opinion that to wait until cavity formation appears before instituting artificial pneumothorax definitely increases the patient's chances of developing this serious complication.

Osler said, "We must collect facts with open-minded watchfulness, unbiased by crotchets or notions; fact on fact, instance on instance, experiment on experiment, facts which fitly joined together by some master who grasps the idea of their relationship may establish a general principle." It is with this thought in mind that we present our small series of patients with air embolism and spontaneous pneumothorax. Many articles have been written on this subject, and we are thoroughly cognizant of the fact that this one contains nothing particularly new, but contributes only to the total number of cases reported in the literature, and may be of slight value to someone at a later time in compiling data and analysing large numbers of cases with the thought of drawing final conclusions.

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THE HEART IN PULMONARY TUBERCULOSIS

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THE subject of the relation of cardiac disease to pulmonary tuberculosis is one which has received rather scant attention in this country, and it may at once be said that there are no startling subjective or objective findings on which to work. Nevertheless, from time to time certain observations have been made which force a conclusion that the response of phthisical patients to treatment may in some measure depend upon the healthy integrity of their cardiovascular system. When we consider the field of thoracic surgery, we realise that a certain proportion of the operative mortality can be ascribed to cardiac failure.

Method of Investigation.

For the purposes of the present investigation each patient admitted to certain wards of the Brompton Hospital during a limited period was included in the series. Exception was made only where the patients were too ill to be moved or where collapse therapy or surgical treatment was expected to be carried out very soon after admission. The investigations included a clinical history and examination, blood pressure determinations, radiographic and electrocardiographic examinations. Most of the patients were suffering from uncomplicated pulmonary tuberculosis, and the results obtained are compared with those of previous observers.

A further small series of fifteen cases in which some form of collapse therapy was carried out is reported.

Physiological Considerations.

The pressure in the pulmonary arteries, although it has not been positively measured in the human subject, has been deduced by analogy from animal experimentation to be of the order of 15 to 20 mm. Hg. The normal capillary bed is very large, and as a result there is a sharp fall in pressure,

so that it is actually believed to be negative in the pulmonary veins. Small changes in the capillary bed have little effect upon the arterial pressure, while experimentally in dogs Moore and Binger[1] have shown that compression of one branch of the pulmonary artery by means of an inflated rubber bag causes no obvious embarrassment, while when the remaining branch is progressively occluded it is only when the lumen is almost entirely obliterated that an increase in respiration rate is observed. Ligation of one branch of the pulmonary artery is promptly followed by the establishment of a considerable collateral circulation through the bronchial arteries, and Schlaepfer[2] has shown that fibrosis only results if at the same time the phrenic nerve is interrupted. Wiggers[3] has pointed out that pressure within the pulmonary artery depends upon four factors: (*a*) The minute output of the heart; (*b*) the pulmonary capillary resistance; (*c*) the viscosity of the blood; (*d*) the resistance of the mitral valve. Valvular disease in association with pulmonary tuberculosis occurs in only some 2 to 3 per cent. of all cases,[4] so that the fourth factor may generally be ignored. Blood viscosity has never yet been adequately investigated, and little is known of the variations which it may undergo in health and in disease. Changes in capillary resistance would only be expected in advanced tuberculosis, and on the basis of the experiments of Moore and Binger would have to be very extensive to cause cardiac embarrassment by mechanical means.

On theoretical grounds, therefore, no cardiac dysfunction resulting from mechanical obstruction is to be expected, and any useful investigation must be directed mainly towards ascertaining whether any intrinsic cardiac degeneration is likely to occur as a result of any factor affecting the myocardium.

It does not appear probable that either nervous or hormonic control of the heart's action is ever of more than transient importance, but, excluding thyrotoxicosis, suprarenal and pituitary disease, there is very little concrete knowledge upon which one can work.

The conclusion reached from a study of physiological data must be that there is no one factor affecting the cardiac function in pulmonary tuberculosis which can be effectively initiated experimentally, and attention must, therefore, in the main be paid to the actual findings in patients suffering from this disease.

Previous Work.

There have been a number of previous investigations whose results have a bearing upon the subject under review. The results obtained by previous workers are analysed here, but only the most essential information has been abstracted.

In 1921 Boas and Mann[5] made an electrocardiographic study of 92 cases. They found that 29 per cent. showed a right ventricular preponderance and 30 per cent. a left ventricular preponderance, the remaining 41 per cent. being normal. They did not find any correlation between the cardiographic changes and the clinical condition of the patients.

In 1928 Simon and Baum[6] studied 250 cases of pulmonary tuberculosis, together with their electrocardiograms. They found a diminution in the average height of the T-waves and found also an association between the low potential in Leads I. and III. and the "dropped heart." They made a very detailed analysis of the voltage of each deflection and of the duration of the various complexes, but even this painstaking labour seems to have brought very little reward. They found right ventricular preponderance in 10 per cent. of the whole series.

In 1929 Anderson[7] reviewed 100 cases, in 58 of which some form of collapse therapy had been employed. No constant cardiographic change was found following the various forms of pulmonary collapse, but he appears to have considered only those cases in which collapse had not been followed by any undesirable result.

In 1930 King and Hansen[8] investigated a series of 100 patients. They measured the cardio-thoracic index and classified their cases as being cardio-asthenic when the index was below 40 per cent., cardio-sthenic when it lay between 40 and 50 per cent., and cardio-hypersthenic when the ratio was greater than 50 per cent. They found 32 per cent. of cases to be asthenic as compared with 21 per cent. of a series of normal control subjects. They found a low potential in one or more leads in 63 per cent. of cases, and the following table shows the correlation between this low potential of the cardio-thoracic indices.

Lead I. : Asthenic, 57 per cent.; sthenic, 38 per cent.
Lead II. : Asthenic and sthenic, 45 per cent.
Lead III. : Less common than in normals.
Total incidence of low potential, 63 per cent.

There are many possible explanations of this finding of a low potential, and among them King and Hansen mentioned:

1. Constitutionally asthenic heart.
2. Decrease in size of the heart in pulmonary tuberculosis.
3. Toxic effect of tuberculosis upon the myocardium.
4. Altered position of the heart.
5. Poor nutrition of the heart.
6. Physical inactivity.

Some of these possible factors are capable of further investigation, but the second one is merely a restatement of the occasional finding of a dropped

heart, a configuration which is probably entirely the result of the shape of the thoracic cavity.

The following data, taken from the results of a number of investigations, appear in the same publication:

	Lead I. (*Per Cent.*).	*Leads I. and III.* (*Per Cent.*).	*Lead III.* (*Per Cent.*).
Asthenic	57	45	19
Sthenic	38	45	71
Hypersthenic	5	10	10

King and Hansen[8] also studied 66 cases in which collapse therapy had been used, and they formed the opinion that such electrocardiographic changes as they found were the result of cardiac displacement, and particularly of pleural and mediastinal adhesions.

In 1933 Heaton studied, and reported in a short article, 304 cases. Of the total, he found 68 (22 per cent.) to show some electrocardiographic abnormality, and of these 49 showed a left axis deviation, while 24 showed right axis deviation.

In the same year Von der Weth[9] published the most extensive monograph which has so far appeared on this subject. He reports the findings in 508 tuberculous patients, but found no correspondence between the clinical and the electrocardiographic data. He pays much attention to minute changes in the various complexes, but it seems doubtful whether some of these changes have any real significance. In a subsequent contribution on the same subject he suggests that three types of cardiac change may be met with in pulmonary tuberculosis:

1. Acute Toxic: Shown by sinus tachycardia.
2. Chronic Toxic: Shown by electrocardiographic evidence of myocardial degeneration.
3. Mechanical: Shown by electrocardiographic changes due to cardiac displacement.

As evidence of myocardial disease Von der Weth placed more importance on prolongation of the P.R. interval than upon the occurrence of a low potential in one or more leads, and he varies his standard of the normal P.R. interval according to the rate of the heart. His most useful contribution would seem to be his suggested classification of the three types of cardiac disorder which may be encountered.

From this review of the literature it will be seen that hitherto no conclusive results have been obtained, and as no previous work seems to have been done on the subject in this country, we were encouraged to test the results obtained in other countries.

The Present Series.

The cases reported are divided into two groups. The first, numbering 47, consists of cases in which no collapse therapy had been undertaken and in which no major pulmonary complication was detected. In the second group, numbering 15, will be found those cases in which some form of collapse therapy had been employed. In 7 cases in the second group electrocardiograms have been taken both before and after thoracoplasty.

The group of cases of uncomplicated pulmonary tuberculosis has been divided into two sections according to whether or not a cardiographic abnormality was found.

Of the 47 cases, 23 showed no deviation from normal, while 24 are thought to show some electrocardiographic abnormality. There were 30 males and 17 females. The majority of the patients were young adults, 74 per cent. being under the age of thirty-five years.

Electrocardiographic Abnormalities.

The 24 cases in which the electrocardiogram deviated from the normal show the following changes:

Left Axis Deviation.—Five cases (9 per cent. approximately) show this abnormality. Each of them was suffering from a moderately extensive tuberculosis, but in none of them was there any great activity at the time of examination. The lowest systolic blood pressure in any of these 5 cases was 110 mm. Hg. One case subsequently had a successful thoracoplasty on account of repeated hæmoptysis.

Right Axis Deviation.—Five cases (9 per cent. approximately) showed this abnormality, and in all of them there was extensive and active bilateral disease. The sputum was positive in all cases, and on clinical grounds the prognosis appeared to be bad. In 2 cases the heart was displaced to the left by fibrosis, and in 1 case it was displaced to the right by a left-sided effusion. In 1 case a systolic blood pressure of 102 mm. Hg and in another of 108 mm. Hg was recorded, but in the remaining 3 cases the pressure was 130 mm. Hg or more.

Low Potential in One or More Leads.—This was found in Lead I. only in 10 cases—*i.e.*, 21 per cent. approximately. In every case the sputum contained tubercle bacilli on one or more occasions, and with only one exception the patients were suffering from extensive bilateral disease. In the one case with unilateral disease there was little clinical evidence of activity, but the heart was displaced to the right by fibrosis and a systolic cardio-respiratory murmur was audible. Three cases in this group showed systolic blood pressure below 110 mm. Hg.

A low potential in Lead III. only was found in 2 cases. The first case showed active unilateral disease, a positive sputum, and was complicated by mild thyrotoxicosis. The blood pressure in this case was normal. The second case showed extensive bilateral disease, positive sputum, and a normal systolic blood pressure. In this case the disease showed little clinical activity.

A low potential in Leads I. and III. occurred in 1 case. This patient had bilateral disease, with displacement of the heart to the left, and showed considerable clinical activity. The systolic blood pressure was occasionally low, but normal readings were generally obtained.

In one case an inverted T-wave in Leads II. and III. was recorded. This patient had very extensive and active disease, with a positive sputum. The blood pressure was normal.

Cases showing Normal Electrocardiograms.

This group consists of 23 cases. In 20 the disease was bilateral, while in 3 it was unilateral. Seven cases showed cardiac displacement, while in 16 the heart was normally placed. A systolic blood pressure below 110 mm. Hg was recorded in 8 cases, only 2 of which showed a cardiac displacement, while 2 others were regarded as having very active disease.

The Heart in Collapse Therapy Cases.

Four cases treated by artificial pneumothorax were investigated. Right pneumothorax was present in 3 cases showing respectively left axis deviation, right axis deviation, and low potential in Lead I. The fourth case with a left-sided artificial pneumothorax showed a low potential in Lead III. and a low systolic blood pressure.

Phrenic evulsion was carried out in 4 cases, and in each of these an electrocardiogram was taken both before and after the operation. Two cases showed a systolic blood pressure below 110 mm. Hg. In one case the electrocardiogram before the operation simulated a slight right axis deviation, and after the operation simulated a left axis deviation, but the changes were not very well marked. The second case showed an initial low potential in Lead III., while after operation the record became normal. In the third case a slight left axis deviation became quite definite after operation. In the fourth case an initial low potential in Lead III. was unaffected. The two cases showing low potential in Lead III. also showed, radiographically, displacement of the heart to the right.

Seven cases were examined electrocardiographically both before and after thoracoplasty. In 1 case only did the character of the record change materially, and in this case a right axis deviation disappeared and a low

potential in Lead I. was subsequently recorded. A systolic blood pressure of 96 was recorded in 1 case. In 2 further cases, right and left axis deviation respectively were recorded and remained unchanged after operation.

Conclusions.

In the series of cases we find that approximately 50 per cent. shows an electrocardiographic abnormality. The severity of the disease was approximately the same whether the electrocardiogram was normal or abnormal. Displacement of the heart produced no constant electrocardiographic change. Collapse therapy produced no predictable change in the electrocardiographic record, and this was particularly noticeable in the patients who had been treated by thoracoplasty.

Only 1 patient in the whole series was found to have a heart murmur, and this was regarded as cardio-respiratory in origin.

Thirty-three per cent. of all the cases showed a systolic blood pressure below 110 mm. Hg, and this was the lower limit of normal which was arbitrarily selected. In only 2 cases out of the 63 studied was the systolic blood pressure below 100 mm. Hg. It is hardly surprising that in a group of patients belonging mainly to the age group twelve to thirty under treatment by rest in bed, and accustomed to their surroundings, should show blood pressures which tend to be near to the lower limit of normal. The frequently repeated statement that a low blood pressure is a significant feature of pulmonary tuberculosis is not acceptable to us as a result of our investigations. We would deprecate the repetition of the statement that a low blood pressure is common in pulmonary tuberculosis without fresh evidence.

In no case was the transverse diameter of the heart greater than 6 inches in a teleradiogram, and the cardiothoracic index was in each case less than 50 per cent.

We have not been able to gain any assistance from detailed examination of the cardiovascular system which would help us in the prognosis or the treatment of pulmonary tuberculosis.

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CLINICAL CASES

INTRACRANIAL TUBERCULOMATA
A RECORD OF FOUR CASES

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Tuberculosis may attack the nervous system in a variety of ways, and from a clinical point of view the manifestations can be divided into three classes:

1. Tuberculosis of the meninges.
2. Tuberculosis of cranial bones and vertebræ.
3. Tuberculosis of the tissues of the brain and spinal cord.

The first two groups are by no means uncommon, and it is to the third group that this record is devoted. Tuberculomata of the brain occurring during the course of a case of tuberculosis are not common, and on that account the following cases are recorded.

Pathology.

Tuberculomata of the brain may occur in any position, but are more commonly located in the subtentorial region.

In size they vary from a small area up to an inch or more in diameter; frequently multiple tumours of small size are present, or the tumour may be solitary and of larger size.

Encapsultation may be present, or the tumour substance blend almost indistinguishably with the brain substance; evidence of caseation may be present in the centre of the tumour. In some cases calcification may be present.

Symptomatology.

The symptoms produced by a tuberculoma are essentially those of any other intracranial tumour, in regard to the signs and symptoms of increased intracranial tension, headache, vomiting and optic neuritis.

Localising signs may be present according to the location of the tumour.

Diagnosis.

The history of the case should be considered in reference to previous or co-existing tuberculosis, and the general methods of examination performed in regard to localisation of intracranial tumours.

Radiological examination may be of value in the event of calcification being present in the tumour, the use of radiological examination of the skull after the introduction of air into the ventricles may assist in localisation.

Examination of the cerebro-spinal fluid may be of assistance, if the tumour is so placed as to give rise to changes in the fluid.

The signs presented may be such that accurate localisation is impracticable, and a record is given of four cases under review.

CASE 1.—J. R. B., male, aged eight years. The history was one of cough, loss of weight and night sweats for four months. No family history of tuberculosis. The signs presented were those of consolidation of the upper lobe of the right lung. The case was admitted to hospital, where a radiological examination confirmed the presence of tuberculous lesion in the right lung; examination of the sputum for tubercle bacilli was negative on five occasions, and examinations of the fæces for tubercle bacilli were negative on five occasions.

In view of the extension of the lesion in the lung after conservative measures had been tried, a right artificial pneumothorax was induced.

An excellent collapse was obtained and this was maintained for one year. Ten days after the last refill the patient commenced to have choreiform movements of the right arm and leg, and was readmitted to hospital.

On admission there were no signs in the nervous system except for the involuntary movements; the patient then complained of occipital headache; no eye symptoms or vomiting. A lumbar puncture was performed, the fluid was under tension, there was no increase of cells, faint excess of globulin, no tubercle bacilli were seen.

Following the lumbar puncture the patient improved, was free from headache, and the movements were almost abolished. Three weeks later the headaches returned and the patient became drowsy, but no vomiting or movements were present; he was then given hypertonic salines.

Ten days later the choreic movements returned, associated with headaches and some dimness of vision.

On examination it was found that the sixth and seventh cranial nerves on the right side showed paresis, the right arm and leg showed choreic movements, inco-ordination, weakness and diminution of reflexes. There was a tendency towards ankle clonus; plantar response normal; no changes noticed in sensory system. The pupils were regular and reacted to light and accommodation; the fundi showed papillœdema, left fundus rather worse than right. Radiological examination of the skull was negative, Wassermann reaction was negative, and examination of the cerebro-spinal

fluid at this time showed globulin test positive, cells 115 per cmm., lymphocytes 60 per cent., polymphs 40 per cent.; a diagnosis was made of subthalamic tuberculoma.

A decompression was then performed with a view to saving the vision, but the patient died two days after operation.

Post-mortem.—The right pleural cavity showed some adhesions and a small quantity of fluid; apart from a small tubercle in the lower part of the right upper lobe, the disease in the lung appeared to be arrested.

Brain.—There was general dilation of the ventricular spaces, more marked on the left side, and on section a tuberculoma the size of a gooseberry was found on the left side involving the lateral wall of the third ventricle.

Microscopy confirmed the presence of tuberculoma and tuberculosis of the right upper lobe of lung.

Case 2.—J. E., male, aged thirty. The patient complained of vomiting and dizziness of seven weeks' duration, followed by cough with muco-purulent expectoration and pain in the right chest. Previous medical history was that of tabes mesenterica, confirmed at operation six years previously; the patient had also suffered from facial lupus.

On examination, the patient showed evidence of pulmonary tuberculosis of the lung, no changes were noticed in the cranial nerves, no abnormal signs detected in the arms. The left leg showed markedly exaggerated knee and ankle reflexes, ankle clonus present and extensor plantar response. There was weakness and loss of tone.

Rombergism was present, with a tendency to fall backwards and to the left; papillœdema was present in left fundus. Radiological examination of the chest confirmed the presence of pulmonary tuberculosis in the right lung. Sputum examination was repeatedly negative; Wassermann reaction negative. Cerebro-spinal fluid had coagulum, lymphocytes 75 per cmm. The patient progressively got worse and died six months after the onset.

Post-mortem.—Tuberculoma left cerebello-pontine angle.

Case 3.—J. K., male, aged fifty-six. The patient had a tuberculous epididymis some years previously and a discharging sinus had persisted. He complained of headache and vomiting seven days prior to being seen. This was followed by diplopia and an attack of vertigo in which he fell and lost consciousness.

On examination the mental state was quite confused, there was wasting of the limbs, exaggeration of the reflexes in the legs and plantar response was extensor, otherwise no other changes were noticed in the nervous system. Fundi showed evidence of papillœdema.

There was evidence of extensive pulmonary tuberculosis and the epididymis was thickened and nodular, with a discharging scrotal sinus.

Post-mortem.—Limited to cranium.

Pia arachnoid exudate over the lateral and superior aspect of right lobe of cerebellum. Œdema of pia.

Brain.—Atrophy of convolutions, ventricles dilated. Two tuberculomata

were present, one in the right cerebellar peduncle about 7 mm. diameter and another in the right cerebellar lobe of similar size.

Tuberculomata of cerebellum and cerebellar peduncles.

CASE 4.—F. D., female, aged nineteen years. The patient complained of headache, and twitchings down the left side of the body, and vomiting of a cerebral type for eleven months prior to operation. The headache was frontal in site and gradually became worse, and for two months the sight became impaired. A diagnosis of a right-sided pararolandic lesion was made, and a right frontal osteoplastic flap was turned down.

Following operation, the patient improved, the fits became less frequent and vision improved with a disappearance of headache and vomiting.

Three months later a submuscular abscess developed above the left knee, sterile pus was aspirated.

Pain began to develop in the region of the lumbar spine, rigidity of the spine and loss of use of the legs appeared. On examination there was paresis of left seventh cranial nerve, vision able to count figures, no other changes in cranial nerves.

Motor System—Left Arm.—Weakness, wasting, tone increased; triceps, biceps, sipunator reflexes exaggerated; tendency to athetosis.

Legs.—Weakness, slight wasting, no increase in tone, no ankle clonus, plantar response flexor, some diminution of knee and ankle reflexes.

Sensory System.—No marked changes except for some impairment of tactile sensibility over the lower part of the body with doubtful level at the umbilicus.

There was a large psoas abscess in the left iliac fossa extending into the left thigh. Tenderness and immobility of the lumbar spine was noted.

There were no other abnormalities detected.

Diagnosis.—Cerebral tuberculoma, tuberculosis of lumbar spine with compression, psoas abscess.

Treatment.—Decompression is advisable to relieve the symptoms of increased intracranial tension, and to save the sight if this is threatened; enucleation is inadvisable, even if the tumour can be located and is accessible, since a fatal meningitis is prone to occur after enucleation. Decompression in itself may be sufficient to tide the patient over the acute stage and give time for healing and fibrosis.

SPONTANEOUS PNEUMOTHORAX ASSOCIATED WITH ABSCESS OF THE LUNG

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Case Report.

During the first half of August, 1936, in spite of rainy weather, the patient, a spinster, aged fifty-one, undertook a walking tour in Yorkshire. She was on several occasions soaked to the skin, with the result that by the end of the holiday she had developed a "chill." On August 12, while hurrying to catch a train, she experienced a sudden severe pain in the left side of the chest and became extremely dyspnœic, but was able to continue her journey home to London. There she was found to be febrile, and took to her bed; expectorant and sedative cough medicines were prescribed and the chest was strapped. Irregular fever, unproductive cough, dyspnœa variable in degree, and continuous pain in the side persisted, and after three weeks her general condition had deteriorated seriously. On September 8 the cough became productive of purulent offensive sputum in considerable amounts, and synchronously there occurred an improvement in other symptoms and a fall in her temperature. The production of sputum, however, again decreased and she became severely ill once more.

The patient was admitted to St. Mary's Hospital on September 12. No other relevant facts in her past history were elicited, and she stated that tuberculosis was unknown in her family. She was desperately ill, orthopnœic and cyanosed, coughed incessantly, producing about 1 ounce of foul sputum daily, and complained of continuous dull pain high in the left axilla. T.=103°, P.=110, R.=36.

Examination revealed the signs of a pneumothorax on the left side, while at the extreme base an area of dullness suggested the presence of some fluid. The coin sound and succussion splash, however, could not be obtained. Coarse râles were uniformly scattered throughout the right lung. Her fingers were not clubbed. Apart from tachycardia and a slight displacement to the right, the heart was not abnormal. There was no vaginal discharge or other evidence of pelvic sepsis, and other systems were clinically normal.

Radiological investigation disclosed the following:

1. A dense opacity is present in the left mid-zone without the hilum, roughly wedge shaped, base at axillary periphery; lower margin somewhat convex downwards, and fairly sharp.

2. The only suggestion of "breaking down" is in a small translucent area at the extreme periphery.

3. Using P.A. and lateral views, the consolidated area is in the posterior and lower part of the left upper lobe; there is probably *also* a small quantity of fluid in the upper part of the interlobar fissure.

4. A small pneumothorax is present with a minimum quantity of basal fluid.

Conclusions.

1. Pneumothorax.

2. Small interlobar and minimal "open" effusion.

3. Probable *chronic lung abscess* left upper lobe posteriorly, with very little breaking down centrally, *or* no free communication with bronchus.

Bacteriological examination of the sputum on two occasions failed to reveal tubercle bacilli, culture showing in each case an almost pure growth of *Streptoccocus viridans*.

Blood culture was negative, and a blood count showed a leucocytosis of 19,000, of which 89 per cent. were polymorphs, without appreciable anæmia.

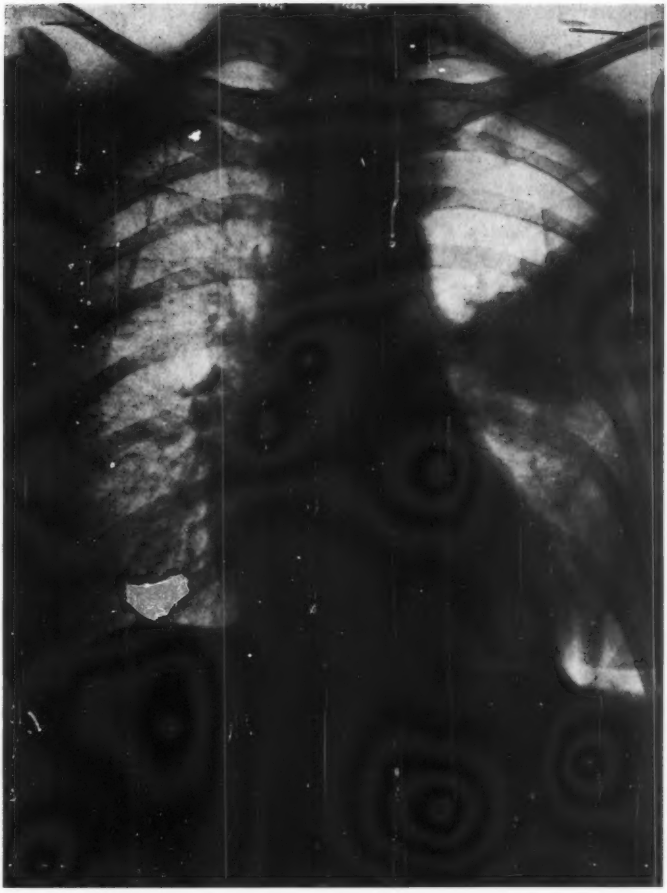
Urine contained a trace of albumen, but no blood or casts.

In view of the severity of the patient's dyspnœa it was tentatively decided to remove some air from the pleural cavity with a pneumothorax apparatus, and accordingly a needle was inserted posteriorly. However, since the intrapleural pressure was found to be $-2-6$ cms. H_2O, it was thought unlikely that the pneumothorax *per se* was contributing greatly to the patient's distress, and since the abscess was peripheral, the removal of air, with a consequent reduction in intrapleural pressure, seemed unwise, and was, therefore, not attempted. Her condition was such that surgical intervention had to be postponed, and medical therapy, including the continuous use of an oxygen tent, was instituted. She failed, however, to respond and died on September 19. A post-mortem examination was not obtained.

Discussion.

Definite proof of the ætiology of the septic process in this patient's lung in the absence of an autopsy cannot be advanced. None the less, on the available evidence it would seem likely that a spontaneous pneumothorax was here associated with a lung abscess, and although this association is mentioned in most of the text-books of medicine, its occurrence is sufficiently rare to justify a record being made of the case.

PLATE X



RADIOGRAM SHOWING LEFT SPONTANEOUS PNEUMOTHORAX WITH LUNG ABSCESS IN THE LEFT UPPER LOBE BREAKING DOWN PERIPHERALLY.

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CONSULTATION

A CASE OF SEVERE DISSEMINATED TUBERCULOSIS IN AN ADULT

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M. A. C. W., aged twenty-four, single, was admitted to the Linford Sanatorium on November 17, 1928. He was the youngest in a family of four. One brother was very ill with pulmonary tuberculosis and two others had died from causes other than tubercle. Till 1927 his health had been excellent, but he was " run down " by overwork before he went to West Africa in December, 1927.

In July, 1928, he had much worry and did considerable journeys on a bicycle. Next month he felt ill and had pain in the left chest and developed influenza, an epidemic of which attacked his station. He was in bed for ten days. His temperature never rose above 100° F. He left hospital feeling unwell and with a cough, but was better after a holiday at 4,000 feet. Shortly after his return to the station he lost his appetite, cough increased, and he began to suffer from indigestion. On October 8 his temperature was found to be 103° F. so he was admitted to hospital and sent back to England. There had been no sputum. On the voyage home his temperature remained raised both morning and evening.

On his arrival at Plymouth he went straight to a nursing home and was seen by the late Dr. Soltau, who diagnosed a left pleural effusion. A few ounces of clear fluid were aspirated. It contained " an excess of cells, all lymphocytes. There has been no sputum, but a good deal of irritable

cough." Temperature was 100° F. a.m. and 101° to 102° p.m. On admission to the Linford Sanatorium he was very thin, anæmic, and weak. He had a constant dry cough but no sputum. Temperature was up to 102·5° p.m.; pulse 96; B.P. 116/60. Constipated. Appetite had improved and was very fair. Physical signs were those of a left-sided pleural effusion. Forty-two ounces of fluid were aspirated and replaced by air. Cough was relieved and evening temperature slightly reduced. Next day he had profuse diarrhœa with considerable abdominal discomfort and distension. This, lasted twenty-four hours. After this his heart returned to normal position and attempts to find fluid or free pleural cavity failed on three successive attempts. Probably the diarrhœa had led to absorption of both fluid and air.

By mid-January he had put on 12 pounds. Evening temperatures were still over 100°. His tongue was clean, appetite and digestion were good, and he was able to read with pleasure. On January 29 he had great abdominal discomfort with distension. Enemata gave some relief, but the abdomen remained distended and tense, even after attacks of discomfort had ceased. No free fluid in the abdomen could be made out, but it was considered probable that intra-abdominal infection was present. He continued to put on weight after the attack had passed. In March 150 c.c. of blood-stained fluid were replaced by air. For two days before this he had complained of some discomfort in the right eye. There was slight congestion of the conjunctiva, with no photophobia. On March 18 slight difference in the size and shape of the pupil was noticed, and atropine drops caused irregular dilatation. Two days later a small yellow spot was seen in the lower and inner quadrant of the iris. The eye was uncomfortable, but not painful; the tension did not appear to be raised. He was seen by Mr. Howard Cook, who found that the spot was behind the iris, pushing it forwards against the posterior corneal surface. Although a tuberculoma seemed the most likely diagnosis, the possibility of a gumma, a cyst, or a neoplasm, either primary or metastatic, were considered. A W.R. was negative.

Mr. Ross, of Bournemouth, was asked to see the case as the mass was increasing in size and the eye was more painful. He thought that the condition was tuberculous and advised rapidly increasing doses of tuberculin. Tuberculin T.R. 1/400,000 was given and produced a slight reaction in the eye: a dose of 1/200,000 produced a decided focal reaction with increase of pain. During this time his appetite was good and he continued to put on weight. Evening temperature was 100° and pulse 96. Appetite was good and bowels kept regular by liquid paraffin. His abdomen was still distended, and at times uncomfortable. There was no

cough or sputum and his nights were undisturbed. About this time he first complained of some stiffness and aching of his wrists, especially the right one. The condition of the eye was not so satisfactory and Mr. Ross had grave doubts as to whether it could be saved. Mr. T. Colley, of Weymouth, saw the patient with Mr. Ross, and it was decided to enucleate the eye should there be no improvement in a week's time. The eye was enucleated on April 10 and was sent to the Royal London Ophthalmic Hospital for examination. A preliminary report was received on June 26, and a final report on July 24.

Report on Eye embedded in Celloidin and stained with Hæmatoxylin and Eosin.

Cornea.—Circumcorneal injection. Distortion of cornea. Contains a mass of exudate which is adherent to the anterior surface of the iris and is undergoing organisation. Fibroblasts, endothelial cells, polymorphs, lymphocytes and a few red cells are entangled in the coagulum. Some of the cells have undergone degenerative changes.

Iris.—The stroma is infiltrated with polymorphs and lymphocytes and a few eosinophil cells scattered through the stroma. There are no aggregations of lymphocytes—no giant cell systems or areas of caseation.

Lens.—Flattened, vacuolated in cortex at posterior pole. Deposit of pigment on anterior capsule.

Ciliary body.—Atrophic cyclitis. Extraversation of leucocytes through pars ciliaris retinæ.

Choroid flattened.

Retina.—Degenerative changes in layer of rods and cones and in ganglion cell layer.

Diagnosis.—Purulent cyclitis.

Removal of the eye did not improve the temperatures, which remained about 100° p.m., but he felt better, except that there was now more pain in the wrists, especially on movement. No swelling was yet noticeable, but there was marked tenderness over the lower two inches of the right ulna. X-rays showed periosteal swelling over the lower part of the ulna. Both wrists were put into splints. Later definite swelling developed over the right carpus and a new focus was found over the left tibia. On June 20, 330 c.c. of clear fluid were aspirated from the left chest.

Early in July swelling was obvious over the left carpus and the swelling over the right ulna softened sufficiently for a few c.c. of pus to be aspirated. T.B.s were not found in smears made from this pus, so the bulk was sent

to the Clinical Research Laboratory for inoculation into guinea pigs. Through August new foci formed over both olecranons, the right instep, the right internal malleolus and over the right mandibular joint.

During this period the patient felt better, appetite was good, and he continued to put on weight. A differential blood count showed:

Polymorphs	75·0 per cent.
Lymphocytes	15·4 per cent.
Large mononuclears	7·6 per cent.
Eosinophils	2·0 per cent.

Sir Henry Gauvain was asked to see the patient on September 29. He had some doubt at first whether the multiple and somewhat symmetrical lesions were of tuberculous origin, and suggested that it might be well if the patient were to go to the Tropical School of Medicine to eliminate any possible tropical infection. The next day we received a report from the Clinical Research that the guinea pigs showed definite and extensive tuberculous lesions. Sir Henry kindly consented to have the patient transferred to his care at Alton. His weight had increased from 8 stones 9¼ pounds soon after admission to 10 stones. Temperature still about 100° in the evenings; pulse 96. His appetite was very fair, but he was liable to abdominal discomfort at times. The abdomen was still distended and tense.

The interesting features of this case are: (1) The widespread distribution of the lesions, commencing first in soft tissues, as reported by Dr. Snowden—*i.e.*, in pleura, abdomen and eye—which progressed to bony lesions as follows, for which the patient was under the care of Sir Henry Gauvain. (2) The initial lack of resistance to tubercle evidenced by the rapid spread of the infection, followed by the marked recuperative power as evidenced by the cessation of development of new lesions and healing of existing lesions.

Perhaps the clearest way of demonstrating these two facts is by tables of the lesions as they occurred and progressed.

Date of Occurrence of Lesions.	*Lesions.*
August, 1928	Pleurisy with effusion.
January, 1929	Peritoneal involvement.
March, 1929	Lesion of right eye.
April, 1929	Lesion of right wrist.
July, 1929	Lesion of left wrist.
August, 1929	Lesion of left olecranon.
	Lesion of right olecranon.
	Lesion of right internal malleolus and foot.
	Lesion of right mandibular joint.

LESIONS PRESENT ON ADMISSION TO MORLAND HALL, OCTOBER 7, 1929.

Site of Lesion.	*Condition on Admission.*	*Subsequent Progress.*
Right wrist	Tuberculous disease of carpus with abscess formation on dorsal and palmar surfaces and sinuses over the lower end of the ulna. X-ray showed caries of the carpal bones extending into the base of the metacarpals and a large focus in the lower end of the ulna.	October: Abscess on the palmar surface of right wrist aspirated. November: Incised. December: Formation of three additional sinuses from abscess on palmar surface of wrist. February, 1930: Abscess on dorsum of hand incised and abscess deep to flexor tendons above wrist incised. March, 1930: Abscess deep to flexor tendons reincised. June, 1930: All sinuses healed.
Left wrist	Tuberculous disease of carpus with abscess on ulnar side of the wrist joint. X-ray showed caries of the carpal bones, particularly of the cuneiform and pisiform.	November, 1929: Abscess on ulnar side of wrist joint draining through two sinuses. April, 1930: Sinuses healed.
Right foot	Tuberculous disease of the ankle joint with abscess extending for a considerable distance upwards among the calf muscles. Sinus present on the inner aspect of the foot. X-ray showed caries of the ankle joint with considerable destruction of astragalus.	October-November, 1929: Abscess in the calf muscles aspirated five times. December, 1929: Abscess counter-drained through small puncture. April, 1930: Sinus healed.
Left foot	Tuberculous disease of the tarsus with a small abscess over the outer aspect of the dorsum of the foot. X-ray showed caries of the tarsus with osteitis of the tibia.	November: Abscess increased in size. December: Abscess incised. April, 1930: Sinus healed. April, 1930: Small subcutaneous abscess formed over extensor tendon of hallux, which was incised. July, 1930: Sinus healed.
Right malar bone ..	Puffy swelling over malar with abscess formation.	November 18: Abscess incised. April, 1930: Sinus healed. Finsen treatment given later to scrofulous skin surrounding the old sinus.
Abdomen	Tuberculous peritonitis with fluid effusion.	December, 1930: Ascites absorbed.
Chest	Thickened pleura at left base; no further fluid formation after admission to Morland Hall.	
Left elbow	History of pain and stiffness but mobility full.	No further involvement.
Right elbow	History of pain and stiffness but mobility full.	No further involvement.

Treatment.

The patient was nursed in a chalet in bed with a back rest. Appropriate splints were applied to the arms and legs and deformity of the joints gradually corrected. Daily dressings of iodoform emulsion were performed to the sinuses, and every effort was made to evacuate the pus in each situation as it formed. This was done by aspiration as long as possible, followed by incision into the abscesses when thinning of the skin occurred, the small sinuses thus formed then being dressed daily and all pus expressed. In each case the pus was at first thick, sanguineous and rapidly forming, gradually changing its character through caseo pus to clear serum. In no case did any secondary infection occur.

The bowels were regulated by petrolagar and phenolphthaleine and a liberal fibre-free diet prescribed. Charcoal was given for flatulence, cod-liver oil and various tonics to improve the appetite. Sun treatment followed by carbon arc light baths was also given, and later local Finsen light treatment to the lupoid area of skin on the right cheek.

General Condition and Progress.

On admission the patient's condition was poor, and in view of the extent of the lesions and the recent rapid development of new foci the prognosis was considered very grave. He was thin, appetite was fair but capricious, bowels irregular, and there were frequent attacks of abdominal flatulence and occasional attacks of vomiting. During October and November the disease remained active in the existing foci with rapid pus formation in connection with each lesion. This necessitated frequent aspirations and incisions to allow free drainage and continual care of sinuses. During these months too there was pyrexia of 100° in the evening with a daily variation of 2° to 3°. No new foci developed, however, and by the end of December most of the abscesses were draining freely with definitely beneficial effects on the general condition. In December, 1929, the evening temperature reached 99° on two evenings only, and in January, 1930, patient was apyrexial. At the same time his appetite improved considerably and he was obviously putting on weight.

In July, 1930, all sinuses were healed.

In April, 1931, patient was allowed to sit up in a chair and gradually commence ambulatory treatment. Exercise was then gradually increased until discharge.

On Discharge, July 20, 1931.

Patient had gained 3 stones 3 pounds 12 ounces while at Morland Hall. Appetite was very good. Bowels regular. He was eating a full normal diet and he was walking well. All sinuses had been firmly healed since July, 1930.

CONDITIONS OF THE LESIONS.

Right wrist: Will hyperextend to one third of a right angle and will flex through 3°. Finger movements slightly limited by adhesions of soft tissues.

Left wrist: Will flex through half a right angle and will extend through half a right angle. All movement of fingers free.

Right foot: Will dorsiflex to a right angle and will extend through 10°.

Left foot: Will dorsiflex to a right angle and will extend through 5°.

Both elbows: Normal.

Right side of face: Scar of old malar abscess still slightly scrofulous; no lupoid patches present.

Chest: Nil abnormal was discernable.

Abdomen: No fluid present. Nil abnormal palpable.

The patient has been seen frequently since discharge. He is now married and leading a normal life. On each occasion he has been seen improvement in mobility of the affected joints has been noted. There have been no new lesions.

MEETINGS OF SOCIETIES

THE TUBERCULOSIS ASSOCIATION

THE new session was opened on November 20, 1936, when Dr. S. Roodhouse Gloyne, after being installed as President, gave the Presidential address entitled "From Consumption to Tuberculosis: A Study of the Disease, Past and Present." Consumption, he said, was a wasting disease of uncertain pathology with a grave prognosis, but now is tuberculosis, a curable disease with a mortality curve steadily falling. He traced the history of our knowledge and early work on the disease. It was remarkable how much the Greeks knew about tuberculosis, and they even practised oleothorax; but it was not until the time of Sylvius that the description of enlarged glands laid the real foundation of the morbid anatomy of tuberculosis. In 1793 Matthew Baillie published the first English text-book on morbid anatomy. The conception of the infectivity, which had been accepted by some from the earliest days, gained further support by Koch's discovery of the tubercle bacillus. This discovery was followed by many problems connected with tuberculin, but in spite of all this work there was some neglect of the factors associated with soil. We still remained ignorant as ever on matters of predisposition to tuberculosis. For example, was the catarrhal child, who returned winter after winter to the clinics, more prone to tuberculosis than his healthy brothers?

Recently we have to thank radiology for an enormous increase in our knowledge. He pointed out, however, that it was a mistake to borrow phraseology from pathology in describing what were, after all, mere shadows, and thought there is an urgent need for a new terminology.

In the evening Mr. G. A. Mason opened a discussion on Thoracoplasty in the Treatment of Pulmonary Tuberculosis.

He said that the ideal case for thoracoplasty was a chronic unilateral one. Lesions with no retractile tendency were unlikely to benefit, and for these cases he thought ligature of the pulmonary artery should have a further trial as it might render certain desperate cases sufficiently fibrotic to justify a subsequent thoracoplasty.

Cases of pyo-pneumothorax were in urgent need of thoracoplasty, but if secondary infection had already occurred a preliminary drainage and cleansing of the pleural cavity was advisable. When there were large cavities the operation should be carried out in several stages and with the greatest caution, as the risk of dissemination after operation was considerable.

Shock might be prevented to a large extent by a careful regulation of the amount and rate of collapse obtained at any one stage. Dissemination of the disease after operation was one of the most frequent complications, especially if there were large cavities or much exudative disease. Another complication was atelectasis from occlusion of a bronchus by secretion, and to prevent these complications coughing and expectoration should be encouraged, and the pain associated with cough could be relieved by strapping the side.

Mr. Mason showed a number of slides and a most interesting cinematograph film to illustrate the stages of the operation.

Dr. J. L. Livingstone said that most of the cases suitable for thoracoplasty should first have been submitted to artificial pneumothorax, which was the ideal method of producing relaxation of lung and collapse of cavities. When pneumothorax was impossible, surgical methods of collapse might be required; but he thought that less drastic operations, aiming at selective collapse and leaving the healthy lung to function, would soon be possible. In his opinion a unilateral cavity, even when dry, should be closed before fibrosis made it impossible to do so.

Mr. J. E. H. Roberts considered it wrong practice to deal with the worst side first. A minimal lesion on one side should be treated by pneumothorax or other means before starting thoracoplasty on the other. Apicolysis should be done with upper thoracoplasty, and the extent of the operation depended on the extent of the lesion. He showed several slides to illustrate extrafascial pneumolysis.

Mr. L. O'Shaughnessy showed some radiographs of Mr. Morriston Davies's cases, showing how complete collapse of the lung had been obtained by rib resection without any extrapleural pneumolysis operation.

JOINT TUBERCULOSIS COUNCIL

At a meeting of the Council held on February 20, 1937, Dr. Lissant Cox, Chief Tuberculosis Officer for Lancashire, retired from the Chairmanship and was succeeded by Dr. S. Vere Pearson, Superintendent of the Mundesley Sanatorium. Dr. Lissant Cox and Dr. D. A. Powell were elected Vice-Chairmen, while Dr. Ernest Ward continues his work as Honorary Secretary. Mr. J. E. H. Roberts has joined the Council as the representative of the Society of Thoracic Surgeons, of which he is President.

Further consideration was given to the report of the Infection Committee on the occurrence and prevention of tuberculosis among nurses which was referred to in the last number of this journal. Attention may be drawn to a paragraph on the hours of duty for nurses which represents the con-

sidered opinion of the Council on this subject: " No conditions of service can be considered satisfactory which permit hours of day or night duty for nurses longer than forty-eight in a week. At the present time the difficulty in obtaining nurses may prevent the general application of this rule, which, however, represents an ideal which could and should be attained. Statistics chiefly compiled from foreign sources indicate that there is an undue incidence of tuberculosis among the nursing staffs of general hospitals which receive tuberculosis patients. In well-conducted sanatoria it appears probable that there is no undue incidence of the disease." Professor Lyle Cummins proposed that the Registrar-General should be asked if he could supply tuberculosis mortality statistics for the nursing profession in general and for all nurses who have been engaged in special institutions for the treatment of tuberculosis. The result of this inquiry will be awaited with interest.

A report on the Blood Sedimentation Test in tuberculosis was presented by Dr. Heaf, and led to an interesting discussion on the value of this test. Professor Lyle Cummins pointed out that as different workers employ different methods it is impossible to formulate exact rules. He urged, however, that a standard citrate solution should always be employed (3·8 per cent.), and that percentage readings recorded should always be those obtained from a column of the same height (100 mm.). The technique employed should always be recorded if comparisons between the figures obtained by different workers are to have any value.

Dr. Burrell urged that we cannot interpret the meaning of the sedimentation reactions in the diseased until we know more of the sedimentation rate in the blood of normal persons. Work at the Brompton Hospital has proved that wide variations occur in the rate of corpuscular fall in the citrated blood of healthy individuals when this is followed in a series of readings made on the same day. The rate may alter during digestion as well as during the disturbances caused by menstruation.

Dr. Tytler presented a comprehensive report on the Cultural Examination of Sputum. It is hoped that this will be printed and circulated at a later date.

Dr. Hawthorne reported that no recent meetings of the Milk Committee had been held. He did not think that any helpful action was possible at the present time, for the vested interests, which are still powerful, are apt to be obstructive, and public interest is more easily aroused in the question of milk as a vehicle of epidemic disease than as a dissemination of tuberculosis.

Dr. Ward, on behalf of the Society of Medical Officers of Health, preferred a request that the Council should advise on the value of sanocrysin and tuberculin in the treatment of tuberculosis. A small Committee was appointed to consider each of these problems. Dr. Burrell consented to act as Convener of the former and Dr. Sutherland of the latter.

REVIEWS OF NEW BOOKS

Orthopædic Surgery. By WALTER MERCER, F.R.C.S. Edin. Second edition. London: Edward Arnold, 1936.

This is a far more ambitious text-book than was the first edition. Thereby it gains, and it loses.

The present reviewer was grateful for the first edition as it was of a size and compactness that made it an ideal book to recommend to the ordinary medical student. The present edition is much larger, and many parts have been rewritten.

It contains much that the student who does not mean to specialise need never know, and this is especially true of the pathology, which is very full and very good.

As in the first edition the general arrangement of the subject into chapters is clearer and better than in most similar textbooks.

Readers of this Journal will be more especially interested in Chapters V. and VI., which deal with tuberculosis of bones and joints, and here again the pathological descriptions are excellent.

The relative incidence, as stated, of bovine and human tubercle, 60 per cent. bovine in Scotland and 70 per cent. in England, is more or less common knowledge and accepted; but one can welcome the definite statement that bone and joint tubercle is only part of a general infection, with an outside, generally glandular, focus, and even more the insistence that a local injury is responsible for the location of the disease in one particular spot.

The present reviewer has considerable experience of the incidence of tubercle in old war wounds, a subject which would repay careful investigation and would amply bear out Mr. Mercer's statement.

We shall also approve the statement that the portion of the long bones especially susceptible to tubercle, the metaphysis, is the same as for osteomyelitis.

Mr. Mercer gives a very good summary of the recent work of Butler and Seddon on the paraplegia of spinal caries and of Fraser's classification of bone tubercle, both of which are outstanding contributions to the specialty.

Perhaps he is a little dogmatic in locating the onset of tuberculous hip invariably to Babcock's area. Primary acetabular disease is not uncommon in England, but we shall agree that the spread to the joint occurs early, and that correct treatment is unaffected thereby.

Taking the book as a whole, it is no longer the relatively small text-book for students, but aims higher.

It is very readable, and perhaps the only adverse comments that can be made are that, in the midst of extremely careful and sound statements, there should be included one or two experimental operations, which should have been published in a less authoritative way.

Medical Classics.

This journal, compiled by Emerson Crosby Kelly, of the Department of Surgery, Albany Medical College, was started in September, 1936. It is proposed to publish ten numbers a year, and so it will appear monthly, except for the holiday months of July and August.

Each issue contains a short biography of the author, with the best obtainable portrait of him; a complete list of his works; selected papers in full, and translated into English if it is in a foreign language.

We have received for review the fifth volume, in which the subject is Theobold Smith, M.D., who was born in New York in 1859. After the biographical introduction, his papers on "Investigations into the Nature, Causation and Prevention of Southern Cattle Fever," and "A Comparative Study of Bovine Tubercle Bacilli and of Human Bacilli from Sputum," are given in full.

In this paper he describes the bovine tubercle bacillus as distinct from the human form. Robert Koch wrote in 1908: "To Theobald Smith of Harvard belongs the credit of having been the first to call attention to certain differences between the tubercle bacilli found in men and in cattle. It was his work which induced me to take up this same study."

The present tendency is to be modern, and to regard anything old-fashioned as useless. A textbook ten years old is considered out of date, and this applies not only to medicine, but to everything in life. One is apt to forget that the modern structure of society has been built up by the experience and work of those who went before us. Over a hundred years ago artificial pneumothorax was described, and yet it was not until the end of last century that it was seriously practised, and only in the last twenty-five years has it become recognised as the greatest method of treatment in cases of pulmonary tuberculosis. It is difficult to avoid the view that it would have been available for the consumptive fifty or more years sooner had proper appreciation of the work of the older physicians been given.

This journal should prove of value not only because of its historical interest, but because it contains original papers by great authorities from whom all may learn something on which to base new methods. It should be included in every medical library, and each volume will live and become more valuable as time passes. It can be obtained from the English agent, Messrs. Baillière, Tindall and Cox, for an annual subscription of 47s. for the ten volumes.